HEALTH, ENVIRONMENT, AND ECONOMIC DEVELOPMENT

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Table of contents

Chapter 1: General Introduction and Overview

1.1. Background

Defined as "development that meets the needs of the present without compromising the ability of future generations to meet their needs" according to the Brundtland Commission (1987), the aim of sustainable development is to provide a long term vision for the society. The different elements that constitute this concept are often organized into three dimensions or pillars: (environmental, economic and social). The environmental one consists in the security of the living and physical environment, including natural resources, while the economic one reflects efficient, stable and sustainable economic growth that is not made at the expense of intergenerational equity. The social dimension is devoted to a good life for all individuals. It includes empowerment, fight against poverty, equity, access to social security, education and good health for all the population.[1]

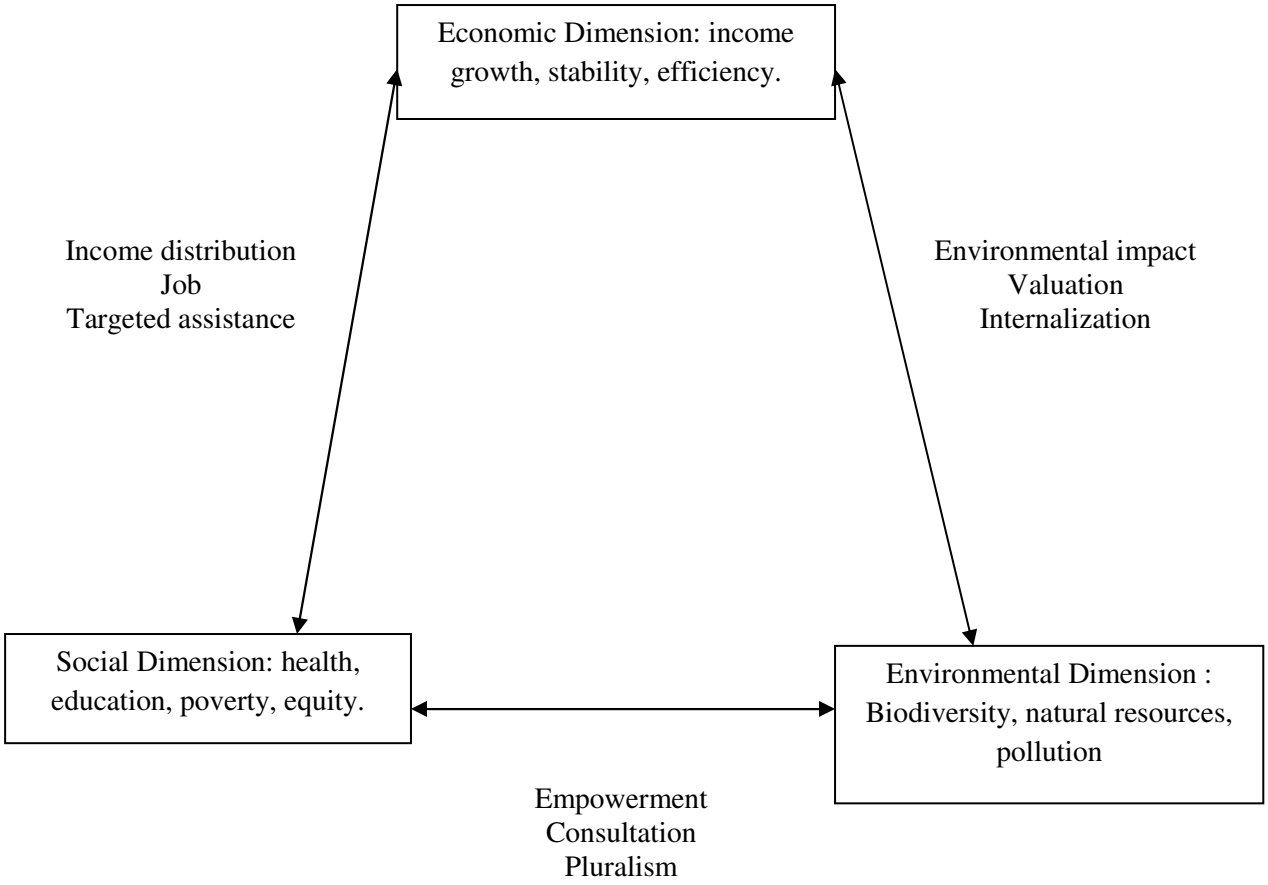
Among these three dimensions of sustainable development, the environmental one is the most known and is even mostly used to represent all the other dimensions. This is probably because, absent from economic field during many years, environmental concerns are more and more present in development strategies since the 1960s. It is nowadays difficult to obtain funding for development projects without quoting environmental advantages and the way environmental degradations caused by the projects are solved. More research papers are published on environmental issues by academics, and environmental preoccupations are at the core of many international meetings. It is one of the eight MDGs (goal 7) adopted by the United Nations in 2000.

[1] The three pillars of Sustainable Development are refered in many United Nations documents since the Brundtland Report, such as the Johannesburg Declaration on Health and Sustainable Development (Munasinghe 1993)

Policy makers, scholars as well as international community are more interested in this concept not only because it is salable (marketable) but also because it plays an important role in the development process and the sustainability of economic development.

The relationships between the three pillars of sustainable development are diversely assessed in the literature, especially when health indicators are used to represent the social dimension (See Figure 1.1). Scholars generally choose two among these three dimensions and investigate their association. Environmental economists are interested in the associations between environmental quality and economic indicators, and usually analyze the link in a bidirectional way.

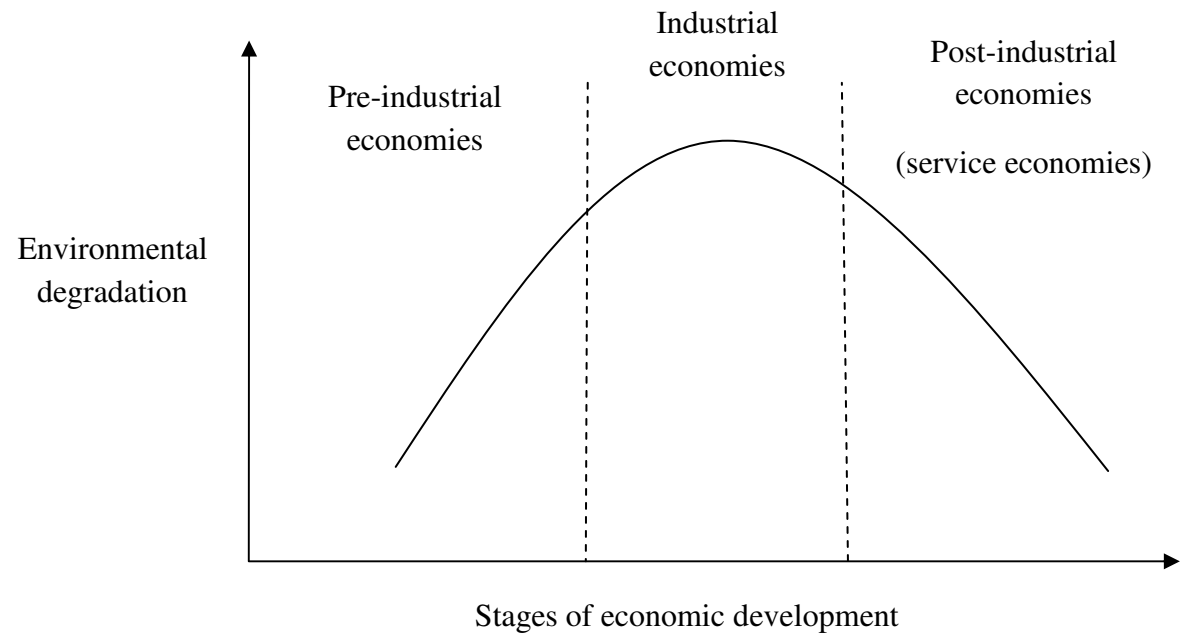
Figure 1. 1: Relationships linking the three pillars of sustainable development.



Source: adapted from Adams, WM. 2001, p. 128.

On the one hand, studies highlight the ways economic activities may affect the quality of environment. Since the early 1990s, empirical works on this field of research have found this relationship between economy and environment as an inverted-U curve called Environmental Kuznets Curve (EKC) (Grossman and Krueger 1993, 1995), analogous to the pattern Kuznets (1955) found between income inequality and economic development. Indeed, according to this hypothesis, environmental degradation tends to rise faster than income growth in the early stages of economic development, then slows down, reaches a turning point and declines with further income growth (Figure 1.2). This hypothesis is not rejected by many studies, even if some authors point out some weakness of these studies and infirm this conclusion (see for instance Carson (2010) or Stern (2004)). It is highlighted in Carson (2010) that the reduced-form nature of the EKC models used in the literature limits the potential policy implications of the results. There is therefore a need for structural models taking into account the likely role of health variables, and the reserse causality linking environment to economic growth in order to propose suitable recommendations to policy makers.

Figure 1. 2: The Environmental Kuznets Curve (relationship between environment and economic development)



Source: Panayotou (2003, pp. 46)

On the other hand, environmental degradation in turn reduces economic performance through its effect on the productivity and the level of human and physical capital. Through its effect on population health, environmental degradation reduces labour supply and labour productivity.

Starting in 1960s, awareness of the environment as important predictor of output growth has steadily increased. Development economists have realized that, their findings based on neoclassical growth models would be incomplete without taking into account environmental concerns (Dagusta & Heal, 1974; Solow, 1974). Since this period many growth model have been developed incorporating environmental issues. Following Panayotou (2000), they can be classified in four main categories: i) optimal growth models, ii) models of the environment as a factor of production, iii) endogenous growth models of environmental degradation and

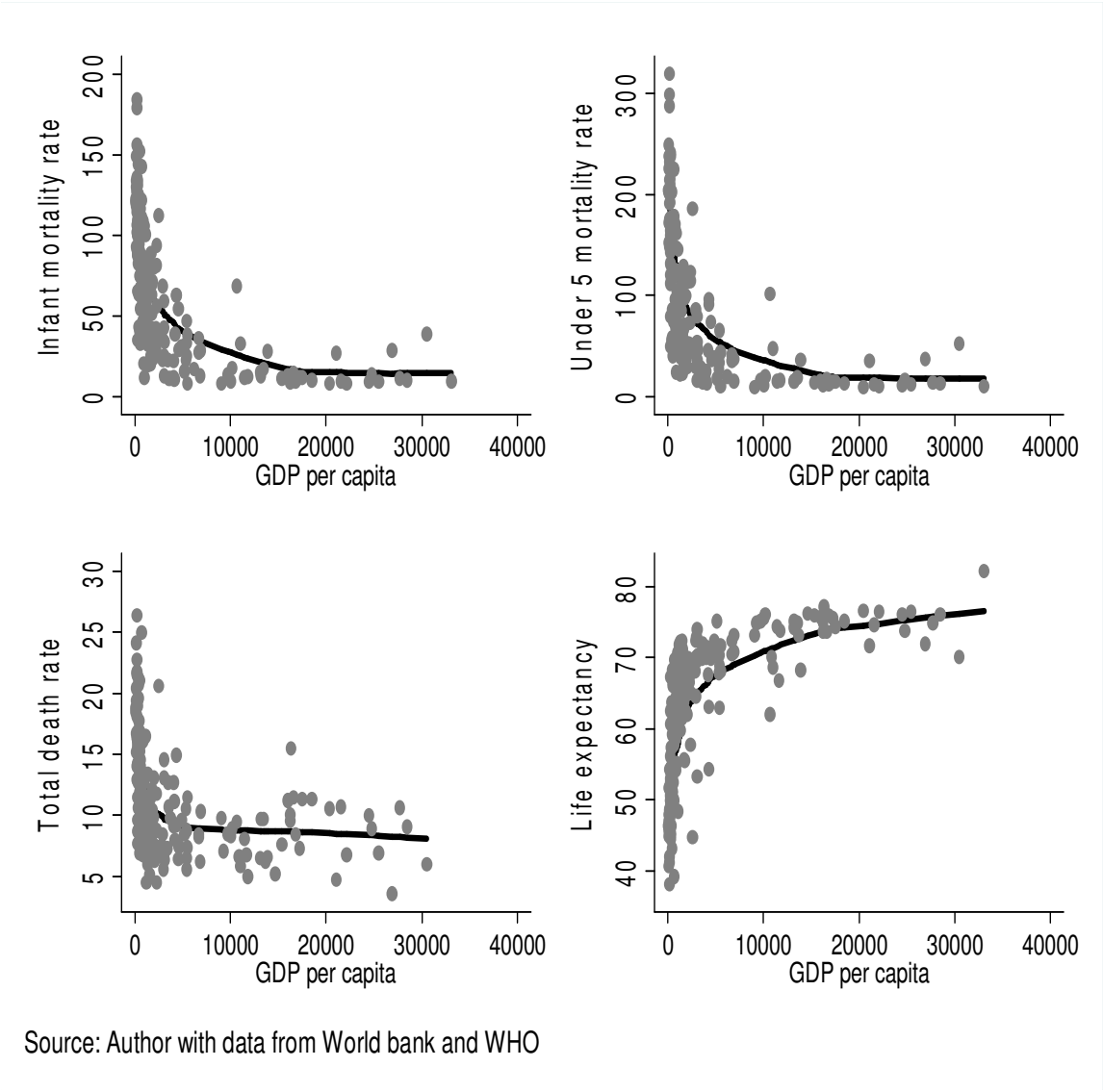
growth, and iv) “other macroeconomic models” of environmental degradation including overlapping generation models, and multisectoral models of growth and the environment in the presence of trade.

Health economists are interested in the relationship between economic indicators and population’s health, and more precisely the effect of health on economic activity (for a literature review, see Audibert, 2010, Schultz, 2010). Besides its direct and immediate effect on people well-being, health status is an important predictor of individual incomes improvements as well as country level economic prosperity (Weil, 2007). Firstly, good health improves the productivity of workers (Hoddinott, 2009) and increases the number of people available as work force in a given population. Secondly, it indirectly improves economic outcome through its effect on education. Improvements in health raise the motivation to attend high level schooling, since the returns to investments in schooling are valuable over a longer working life. Healthier students also have more attendance and higher cognitive functioning, and thus receive a better education for a given level of schooling (Thuilliez, 2009). Moreover, good health encourages more saving and thus investment and per capita productive capital (Chakraborty, 2004).

Figure 1.3 highlights the association between health outcomes and Gross Domestic Product (GDP) per capita respectively when health is measured by infant mortality rate, under five mortality rate, crude death rate and life expectancy.

All these graphs confirm the association between GDP per capita and health status explained above, since health outcomes improve with the level of income. This positive and concave relationship is known in health economics as the Preston curve (Deaton, 2003; Preston 1975).

Figure 1. 3: Link between GDP per capita and health outcomes

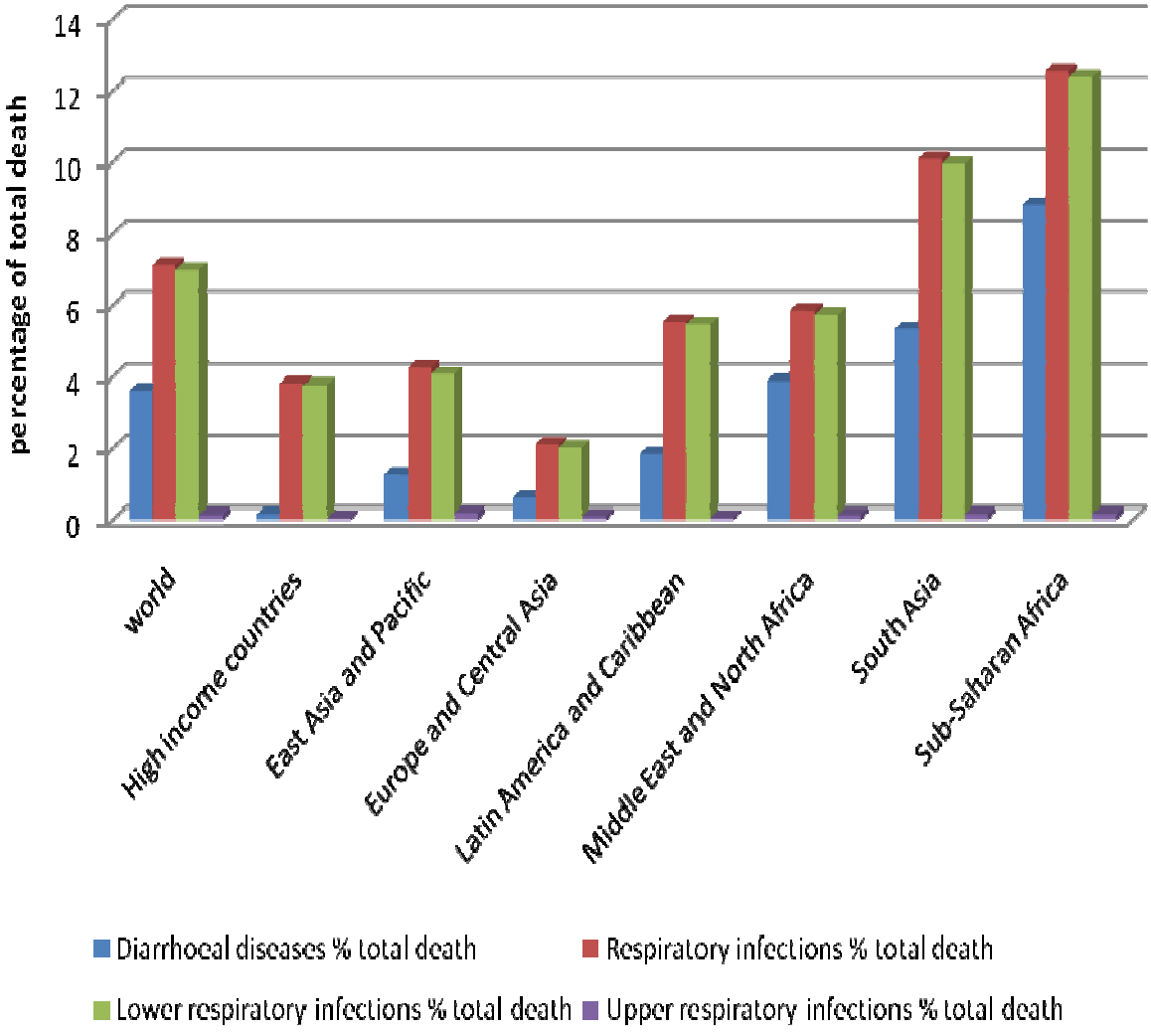


Source: Author with data from World bank and WHO

From these two empirical relationships (environment-economic growth, and health-economic activity), we can infer the existence of the obvious relationship between population's health, and environmental degradation. Figure 1.4 shows the number of deaths from some environmental infections as percentage of total death in 2004 around the world. From this

graph, it appears clearly that the poorest regions such as Sub-Saharan Africa and South Asia suffer more from environmental degradation.

Figure 1. 4: Death from environmental disease as percentage of total death in 2004



Source : Author's construction with data from WHO.

The relationships between these three pillars (economy, social, and environment) remain less studied and explored despite the important challenges and policy implications it may arouse for developing countries. In fact, from our knowledge, existing empirical studies do not investigate simultaneously the link among the three pillars. Health status may play important role in the relation linking environmental degradation and economic preoccupations. Similarly, physical environment variations are not negligible in the association between economy and health. Moreover, the relationships among these three dimensions may imply important consequences for poor countries. This raises the necessity to investigate these complex relationships and its consequences for these countries. This dissertation aims to analyze theoretically as well as empirically the association among population health, environmental degradation and economic development, its consequences for developing countries, and some effective policy responses. Before examining in details all these issues in the following chapters, let explore the outline and main results of this dissertation.

1.2. Outline and main results

This dissertation extends some previous important results on health and the environment by establishing a link between the three dimensions of sustainable development. It is organized in two main parts which themselves embed two chapters. The first part (Chapters 2 and 3) is devoted to the relationship between the three pillars by focusing on a particular aspect of each of them, namely, health (social dimension), pollution (environmental dimension), and income inequalities (economic dimension). It focuses on health outputs of development process by introducing inequality variables in the established link between health and environment; taking two perspectives (see Figure 1.5).

In the literature, income inequality is theoretically and empirically found to have a negative effect on population's health through four main mechanisms: absolute income, relative income, psychosocial, and Neo-materialism hypotheses. Despite the large debate on income inequality as a likely determinant of environmental degradation on the one hand, and the literarature on the effect of pollution on health on the other hand (see Figure 1.5), no study from our knowledge is interested in the probably role of pollution in the relationship linking income distribution and population health. We bridge this gap by investigating how environmental degradation could be considered as an additional channel through which income inequality affects infant and child mortality (Chapter 2).

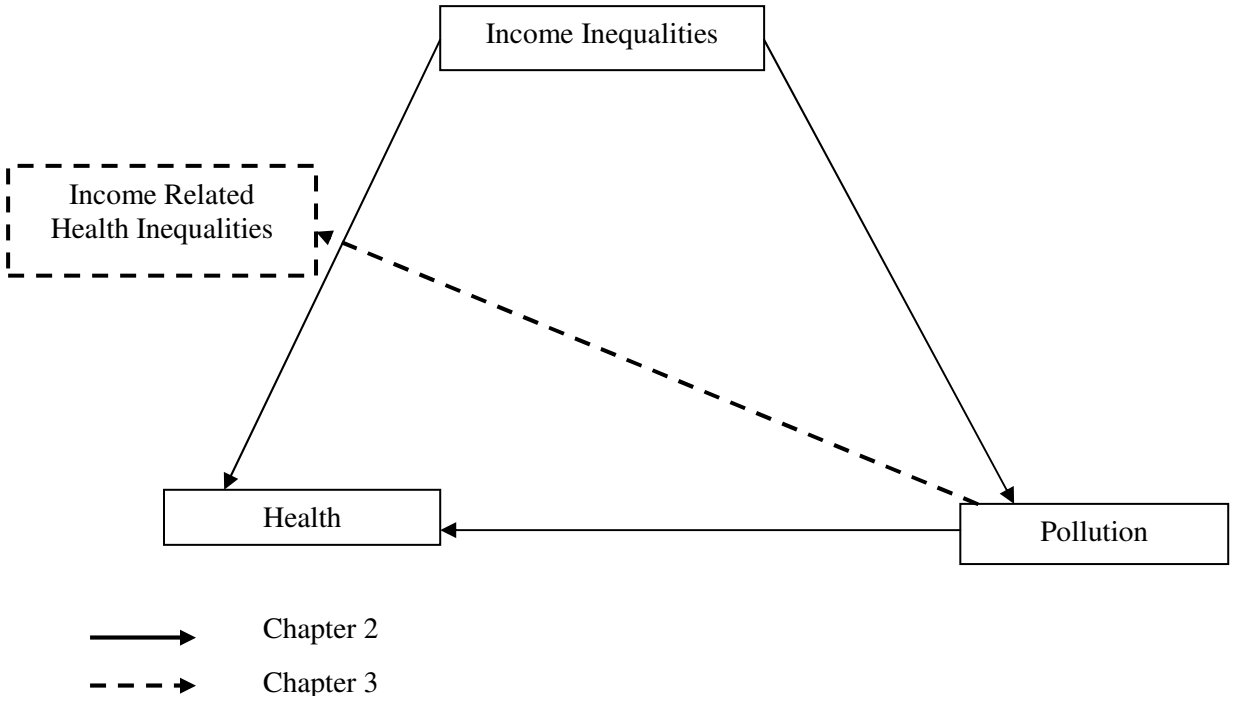
The theoretical and econometric analyses show that income inequalities negatively affect environmental quality, and environment degradation worsens population's health. This confirms that environment quality is an important channel through which income inequalities affect population health. These results hold for air pollution indicators (PM10 and SO2) and water pollution indicator (BOD).

Besides income distribution concern, intra country health inequalities represent an important issue largely approached in the health economics literature. Indeed, in health and environmental economics literature, many studies have assessed the association between environmental degradation and health outcomes. Chapter 3 goes beyond this literature by focusing on health inequalities both between and within developing countries.

Theoretically, it is argued that differential in exposition to air pollution among income classes, prevention ability against health effect of environment degradation, capacity to respond to disease caused by pollutants and susceptibility of some groups to air pollution effect are sufficient to expect a positive link between air pollution and income related health inequality. Furthermore, in democratic countries, this heterogeneity in the health effect of pollution may

be reduced since good institutions favour universal health policy issues, information and advices about hygiene and health practices, and health infrastructures building. Using quintile data from surveys and measuring health inequality as the distribution of health outcome among income quintiles, our econometric results show that sulphur dioxide emission (SO_2) and particulate matter (PM_{10}) are in part responsible for the large disparities in infant and child mortalities between and within developing countries. In addition, we found that democratic institutions play the role of social protection by mitigating this effect for the poorest income classes and reducing the health inequality it provokes.

Figure 1. 5: Health, Environment, and Inequalities.



Source: Author's construction based on Adams, WM. 2001, p. 128.

The first part allowed us to understand how health and environment are linked to inequalities. The second part of the dissertation focuses on growth output of the development process,

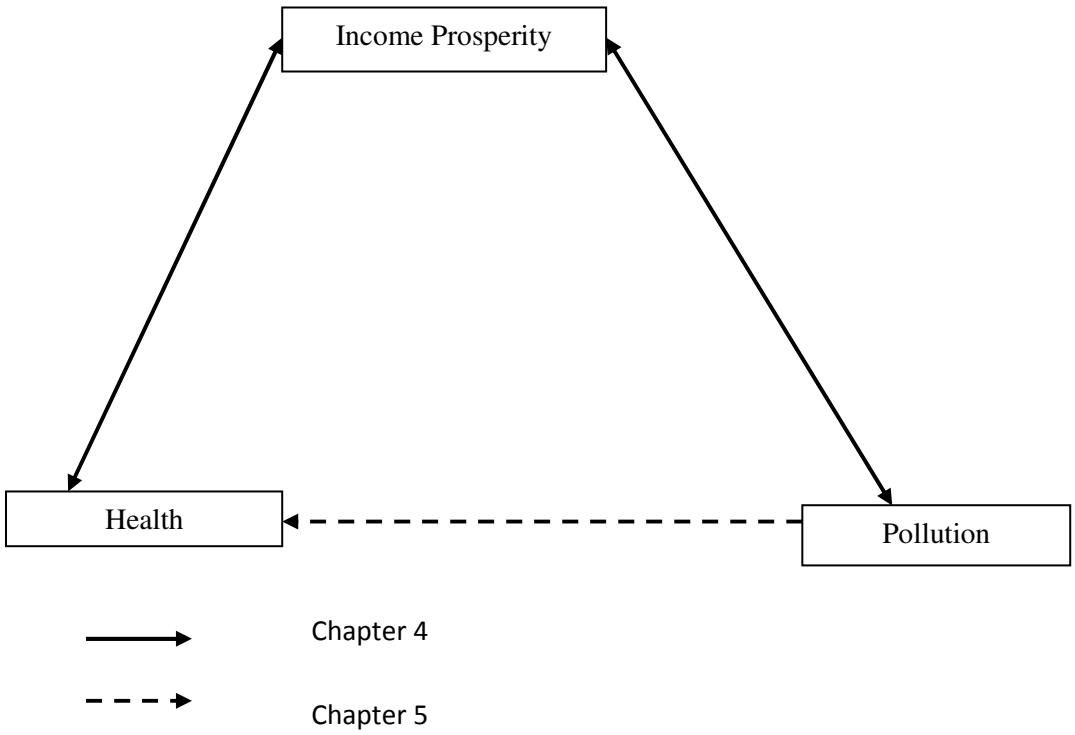
taking into account the quality of the environment and population health. Also based on the three pillars of sustainable development and constituted of two chapters (chapters 4 and 5), it is particularly interested in the inversed-U shaped relationship between economic development and environmental degradation. It investigates through the association between economic development, health, and environment, the risk of weak economic convergence because of bad health and environmental degradation in poor countries (see Figure 1.6).

The assessment of the role played by health outcome on economic growth arouses at least two important problems. First, the direction of the causality is often questioned and becomes subject of a vigorous debate. For some authors, diseases or poor health have contributed to poor growth performances especially in low-income countries. For others, the effect of health on growth is relatively small, even if one considers that investments which could improve health should be done. Besides occurred biases in health measurement. Indeed, commonly used health indicators in macroeconomic studies (e.g. life expectancy, infant mortality or prevalence rates for specific diseases such as malaria or HIV/AIDS) imperfectly represent the global health status of populations. Health is rather a complex notion and includes several dimensions which concern fatal (deaths) and non-fatal issues (prevalence and severity of cases) of illness. The effects of health on economic growth vary accordingly with the health indicator used and the countries included in the analyses. The Chapter 4 (part II) analyze this issue by assessing the effect of a global health indicator on growth, the so-called disability-adjusted life year (DALY) that was proposed by the World Bank and the WHO in 1993. Growth convergence equations are run on 159 countries over the 1999-2004' period, where the potential endogeneity of the health indicator is dealt for. The negative effect of poor health on economic growth is not rejected thus reinforcing the importance of MDGs.

The Chapter 5 extends these analyses, and studies economic convergence with traditional health indicators taking into account the role of the environment. It focuses on the

interrelationships between health, environment, and economic growth, and studies the implications of this relationship for economic convergence through theoretical and empirical models. Environmental variables are introduced in the augmented Solow growth model in order to show the consequences of environmental degradation in terms of economic convergence. To empirically assess these issues, we proceeded to an econometric analysis through three equations: a growth equation including environmental variable, a health equation and an environment equation. We found that environmental degradation affects negatively economic activity and reduces the ability of poor countries to reach developed ones economically. Moreover, as pollution has a negative effect on health, the effect of environment degradation on economic growth is reinforced. This implies that environmental quality could be considered as a constraint for economic convergence.

Figure 1. 6: Health, Environment, and Economic Growth.



Source: Author's construction based on Adams, WM. 2001, p. 128

Part I: health, environment and inequalities

Introduction

Based on the development challenges faced by developing countries, the United Nations established on September 2000 eight measurable development goals to be achieved by these countries by 2015. Environmental sustainability and population's health, already recognized as two important pillars of sustainable development, constitute together half (four out of eight) of these goals (goals 4, 5, 6, and 7). The achievement of these two objectives requires the knowledge of the factors that determine them, but it is also important to find the relationship linking them.

Theoretical and empirical works on the association between environmental degradation and population's health generally find consensual results. It is shown that the direct and obvious consequence of environmental degradation is the deterioration of population's health (Pearce and Warford 1993).

On the other hand, income distribution is considered in the literature to be a determinant of both environmental degradation and population health. Some authors showed that an increase in income inequality degrades physical environment (Boyce, 1994; Ravallion et al., 2000), while others highlight its effect in terms of damage to population's health (Deaton, 2003; Babones, 2008).

Despite the potential relationship between health, environment, and inequalities, it did not arouse much interest to researchers, especially for developing countries. The first part of the dissertation bridges this gap by analyzing the relationship between health, environment and inequalities. It is subdivided into two chapters (chapters 2 and 3). The first chapter entitled "Impact of Income Inequality on Health: Does Environment Quality Matter?" extends the literature on the association between the distribution of income and health status. It

investigates theoretically and empirically, how environmental degradation could be considered as an additional channel through which income inequality affects infant and child mortality.

Then, chapter 3 entitled “Do Political Institutions protect the poor? Intra Countries Health Inequalities and Air Pollution in Developing Countries”, analyses the association between the degradation of air quality (measured by sulphur dioxide emission per capita (SO_2) and particulate matter less than 10 µm aerodynamic diameter (PM10)), and health inequality between and within developing countries. It explores also the role of political institutions in this relationship.

It is globally found that environmental variables play important role in the relation linking health and inequalities. In fact, the effect of income distribution on health is partly channeled by pollution, and environmental degradation exacerbates income related health inequality.

Chapter 2: Impact of Income Inequality on Health: Does Environment Quality Matter?[2]

[2] A version of this chapter was published under the reference: Drabo, A., 2011. Impact of income inequality on health: does environment quality matter? ***Environment and Planning A***, 43(1), 146-165.

2.1. Introduction

Population health is an important economic concern for many developing countries. It plays a crucial role in the development process, since it constitutes a component of investment in human capital and workforce is the most abundant production factor in these countries. It constitutes also a major preoccupation for the international community, especially when it is considered as a public good. The importance given to health status could be illustrated through its relatively high weight among the Millennium Development Goals (MDGs), of which three are related to health preoccupations. It is therefore important to know the factors that influence population health in order to undertake suitable economic policy.

Rodgers (1979) is one of the first economists to consider income distribution as a determinant of health outcomes. He shows that income inequality influences health status not only in developed countries, but also in developing countries, opening the debate about the association between income distribution and health. Wilkinson (1992) reopens the debate showing that income inequality is an essential determinant of health status in eleven industrialized countries. Even though major part of the studies on this topic confirm the negative effect of inequality on health, some authors reject this hypothesis and show that high inequality may be indifferent to health status or improve it (Pampel and Pellai 1986 ; Mellor and Mylio, 2001; Deaton, 2003).

All the mechanisms through which income distribution impacts health status developed in the literature show that an increase in income inequality worsens population health. These mechanisms rely on the absolute and relative income hypothesis, psychosocial hypothesis and neo-materialism hypothesis as well (Mayer and Sarin, 2005). In this paper we add the environment as another mechanism through which income distribution could affect health status. During the past fifteen years, with the emergence of environmental concerns, many

studies examine the association between income inequality and natural environment quality. But they found different results. On the one hand, some authors show that more inequality may improve environmental quality (Scruggs, 1998; Ravallion et al., 2000). On the other hand, other studies underline the negative impact of inequality on environmental quality (Boyce, 1994; Torras & Boyce, 1998). If environmental quality is degraded by an increase in inequality, it may be a channel that reinforces the negative effect of the other mechanisms. But if it is improved by an increase in inequality, it maybe a mechanism that mitigates or cancels the negative effect predicted by the other mechanisms and justify discrepancies between the findings.

Our results show theoretically and empirically that an increase in income inequality is detrimental to the environment and that environmental quality is itself an important contributor to health status. That is, an increase in inequality worsens population's health via environmental degradation.

The rest of this chapter is organized in four sections. Section 2 reviews the literature on the association between income distribution, environmental degradation and population's health. In this section we explain why and how income inequality affects health before introducing the arguments that defend the association between income distribution and environmental quality. In section 3, we investigate empirically the effects of income distribution on health via environment quality. The last section concludes.

2.2. Literature review

The relationship between income inequality and population health has been investigated by many macroeconomic studies during the past 15 years. Scholars examine how and why income inequality affects health theoretically and empirically within and between nations. We will first review the traditional mechanisms, namely the ways income distribution affects population's health already developed in the literature. Then, we will explain how income inequality impacts health through environmental degradation.

2.2.1. Traditional effects of income inequality on health

Theoretically, four mechanisms are underlined, through which income inequality can harm directly population health (Mayer & Sarin, 2005).

The first mechanism is the absolute income hypothesis. In fact, income may be an important determinant of population health, since it allows them to buy better nutrition or medical care or reduces their stress. If the relationship between an individual income level and its health status is linear, an extra unit of income will have the same effect on health regardless of whether it goes to the rich or to the poor. In this case taking a unit of income from the rich and giving it to the poor will lower health status among the rich and raise it among the poor by exactly equal amounts, leaving the global health unchanged. The reality is that standard economic models predict that the health gains from an extra unit of income should diminish as income rises (Preston, 1975; Laporte, 2002; Deaton, 2003; Backlund et al., 1996; Babones, 2008), in other words, health should be a concave function of income. That is, a transfer of a unit of income from the rich to the poor might improve aggregate population's health status.

The second mechanism developed in the literature is the relative income hypothesis. The effect of economic inequality is likely to depend to some extent on the geographic proximity of the rich to the poor (Mayer & Sarin, 2005). In fact, if people assess their income by comparing themselves to their neighbours, the income of others can affect their health. The chronic stress provoked by this comparison may lower resistance to some diseases and cause premature death. For Wilkinson (1997), if individuals evaluate their well-being by comparing themselves to others with more income than themselves, increases in economic inequality will engender low control, insecurity, and loss of self esteem.

According to Subramanian et al. (2002, p.289), these two first hypotheses are not really independent.

The third way developed in the literature through which income inequality may worsen population health is psychosocial hypothesis. Inequality can impact health through social comparisons by reducing social capital, trust and efficacy (Kawachi & Kennedy, 1997; Bobak et al., 2000). According to Wilkinson (1996), income inequality worsens health because a low ranking in the social hierarchy produces negative emotions such as shame and distrust that lead to worse health via neuro-endocrine mechanisms and stress-induced behaviors such as smoking, excessive drinking, taking dangerous drugs, and other risky activities (Mayer & Sarin, 2005). Lynch et al. (2001) found weak associations between a variety of measures of the psychosocial environment, (distrust, belonging to organizations, volunteering, and efficacy), and infant mortality, but they found that economic inequality is strongly related to infant deaths.

Neo-materialism hypothesis is the fourth mechanism through which income inequality may harm health status. According to some authors defending this idea, income inequality affects health mainly through its effect on the level and the distribution of material resources

(Coburn, 2000 and Lynch, 2000). This argument suggests that poor health could be the consequence of an increase in income inequality that reduces state spending on medical care, goods and services for the poor.

If theoretically, all the arguments found in the literature indicate a negative impact of income inequality on health status, empirical findings are far from a consensus (Subramanian and Kawachi, 2003, 2004; Lynch et al., 2004). Lynch et al. (2004) review 98 aggregate and multilevel studies to examine the associations between income inequality and health. They conclude that overall, there seems to be little support for the idea that income inequality is a major, generalizable determinant of population health differences within or between rich countries. Income inequality may, however, directly influence some health outcomes, such as homicide in some contexts. Mayer & Sarin (2005) review ten studies that use cross-sectional data to estimate the association between economic inequality and infant mortality. Eight of these ten use cross-national data and produce eleven estimates. Nine find that more unequal countries have higher infant mortality rates, and two (Pampel & Pellai, 1986; Mellor& Milyo, 2001) find that more unequal countries have lower infant mortality rates than countries with less inequality. Wilkinson & Pickett (2006) compiled one 168 analyses in 155 papers reporting research findings on the association between income distribution and population health, and classified them according to how far their findings supported the hypothesis that greater income differences are associated with lower standards of population health. They find that for 87 of these studies the coefficient of income inequality is always statistically significant with the correct sign. 44 present mixed results and 37 no significant coefficient. They explain the divergence of empirical findings by the size of area, choice of control variables and don't find any explanation for some international studies.

It is worth noting that theoretical works on income inequality and health are mainly based on individual or household considerations whereas empirical studies are generally interested in more aggregate levels (state or country level).

We argue here that in addition to the traditional mechanisms through which income inequality degrades population's health, there exists at least another channel through which income inequality may affect health, namely environmental quality.

2.2.2 Income inequality and environment

A large body of research has reported strong associations between income inequality and environmental degradation: some theoretical arguments are used to explain how income inequality may improve environmental quality (Scruggs, 1998; Ravallion et al., 2000; Heering et al., 2001) while other scholars defend the detrimental effect of increasing inequalities on environment (Boyce, 1994; Torras & Boyce, 1998).

For those supporting the environmental improvement effect, income inequality can increase environment protection through individual preference toward environmental quality. In fact, for a given level of average income, greater inequality means not only higher incomes for the rich, but also lower incomes for the poor. Assuming that the income elasticity of demand for environmental quality is positive[3], and taking a unit of income from the poor and giving it to the rich increases the demand for environmental quality of the rich, but at the same time it decreases the demand of the poor. The net effect on environmental quality depends on whether the demand-income relation is linear, concave or convex (Scruggs, 1998; Boyce, 2003). If this relation is linear, the transfer will not have any effect on environmental quality

[3] This supposes that environmental quality is a normal good

since an extra unit of income will have the same effect on environmental demand regardless of whether it goes to the rich or to the poor. If the environmental demand is linked to income by a convex (concave) relation, the transfer of income from the poor to the rich will increase (decrease) environmental demand.

It is more convincing to assume that the wealthiest prefer more environmental quality than the poor for many reasons. First, economic theories suggest that the rich prefer less environmental degradation than the poor. This may be due to the fact that environmental quality is a superior good of which demand increases faster than income (Baumol and Oates, 1988). This is one of the explanations behind the environmental Kuznets Curve (EKC) hypothesis (Grossman & Krueger, 1995). As argued by Scruggs (1998), greater demand for environmental protection among the wealthiest is also expected to result in a greater willingness and ability to pay for more environmental protection. In addition, wealth increases individuals' concern for the future, maybe because they expect higher life expectancies than the poorest or because it increases their concern for their children in the future. Another reason to explain why rich prefer more environmental quality is that environmental protests are usually composed of middle and upper classes, not the poor (Dalton, 1994).

Income inequality can also reduce environmental degradation through the marginal propensity to emit (MPE) as argued by Ravallion et al. (2000). According to these authors, each individual has an implicit demand function for carbon emissions since the consumption of almost every good implies some emissions either directly via consumption or indirectly via its own production. They call marginal propensity to emit (MPE) the derivative of this implicit demand function with respect to income. If poor people have a higher (lower) MPE than rich ones, a redistribution policy that reduces inequalities will increase (decrease) carbon emissions. One can assume that the poorests have higher MPE than wealthiests, first because less emission goods need high technology and are thus generally expensive. Therefore, the

poorest cannot afford it. In addition, poor tend to use energy less efficiently than the rich, which entails a higher MPE (Ravallion et al., 2000).

If these arguments predict an improvement of environment quality channelled by income inequality, it is also largely argued by some authors that inequality may degrade environment rather than improving it.

Boyce (1994) is the first author to examine how income inequalities affect environmental degradation. He supports the hypothesis that greater inequality may increase environmental degradation and this for two reasons. First, he argues that a greater inequality increases the rate of environmental time preference for both poor and rich. In fact, when inequality increases, the poor tend to overexploit natural capital, because they perceive it as the only resource they have and the only source of income that can help them secure their survival. This environmental effect of poverty is largely emphasized in the literature (Reardon and Vosti, 1995; and known as "poverty environment thesis" since the Brundtland (1987) report. This hypothesis suggests that the poor are both the agents and victims of environmental degradation. In addition to the poverty effect, economic inequality often provokes political instability and risks of revolts. This leads rich people to prefer a policy that consists in exploiting the environment and investing the returns abroad rather than investing in the protection of local natural resources. Therefore, for Boyce an increase in inequality induces both rich and poor to degrade more their own environment. The second argument put forward concerns the power of the richest. Boyce (1994) argues that in a society with greater inequality, rich people are likely to have large political power and can heavily influence decisions on environmentally damaging projects. Such decisions are based on the competition between those who benefit from the environmentally degrading action and those who bear the costs of it. Boyce (1994) argues that rich people are generally the winners, while poor people tend to be the losers of the investments that have an ecological impact. Therefore, economic

inequality favours the implementation of environmentally damaging projects and investments since it "reinforces the power of the rich to impose environmental costs on the poor" (Ravallion et al., 2000, p.656). Scruggs (1998) has criticized the hypotheses supported by Boyce. He states that the influence via cost-benefit analysis is based on two wrong assumptions. First, according to Scruggs, "Evidence indicates that better off members of society tend to have higher environmental concern than those with lower income" (Scruggs, 1998, p.260). Moreover Boyce (1994) assumes that a democratic social choice criterion leads to higher environmental protection than a non-democratic decision process (i.e. a power-weighted social decision rule), while evidence suggests that this is not necessarily true.

Another theoretical argument to explain why more inequality leads to more degradation is developed by Borghesi (2000). He argues that "much of the theoretical environmental literature has stressed the need of cooperative solutions to environmental problems. In an unequal society this is more difficult to achieve than in an equal society since there are generally more conflicts among the political agents (government, trade unions, lobbies etc...) on many social issues. In this sense, greater inequality can contribute to increase environmental degradation" (Borghesi, 2000 p. 4).

In addition to these arguments, some theoretical model supports the environmental degrading effect of income inequality. It is the case of Magnani (2000) who examines the impact of income distribution on public research and development expenditures for environmental protection. Through a model in which social decisions are determined by the preferences of the median voter, she hypothesizes that income inequality reduces pro-environmental public spending due to a "relative income effect," and higher inequality shifts the preferences of those with below-average income in favour of greater consumption of private goods and lower expenditure on environmental public goods.

Marsiliani and Renström (2000) have also recently investigated how income distribution affects political decisions on environmental protection. Through an overlapping-generations model, they show that the higher the level of inequality in terms of median-mean distance, the lower the pollution tax set by a majority elected representative. Therefore, inequality induces redistribution policies that distort economic decisions and lower production. Inequality may be negatively correlated with environmental protection as it leads to less stringent environmental policies.

It is a priori difficult to predict the effect of income distribution on environment quality theoretically even though degrading effect seems in our viewpoint more convincing. Let us see empirical findings.

Empirically studie on the relation between income distribution and environment quality are quite not consensual. In Appendix 2.1, we report nine important papers and thirty one studies on the association between income distribution and environment quality. Among these studies, ten conclude that inequality improves environment quality, nine find the opposite conclusion and twelve don't find any significant association. Let explore some of them.

Scruggs (1998) performs two cross-country empirical analyses to assess the effect of income inequality on the environment through pooled models. In the first one, four different pollutants (sulphur dioxide, particulate matter, fecal coliform and dissolved oxygen) are used as dependant variable in a panel of 22 up to 29 countries. The second investigation examines the impact of several variables on a composite index of environmental quality in a panel of 17 OECD countries. This index is constructed by combining five pollution indicators.

In the first case, he finds conflicting results: greater inequality improves environmental quality for one environmental indicator (particulates), whereas the opposite holds for the other indicator (dissolved oxygen). For the other indicators (sulphur dioxide, fecal coliform), the

coefficients are not statistically significant. In the second analysis, income inequality decreases environmental degradation.

Through a panel of 42 countries in the period 1975-92, Ravallion et al. (2000) first estimate CO2 emissions as a cubic function of average per capita income and of population and time trend. They estimate their equation with fixed effect model and simple pooled model using ordinary least squares. They conclude that higher inequality within countries reduces carbon emissions. However, the impact of income distribution on the environment decreases at higher average incomes.

Borghesi (2000) performs an empirical analysis similar to that of Ravallion et al. (2000). He uses CO2 per capita as environmental variable and Gini from Deninger and Squire as income inequality indicator with a panel of 37 countries from 1988-1995. In the pooled OLS model, an increase in inequality lowers CO2 emissions, whereas it does not have a significant impact on CO2 emissions according to the FE model.

Magnani (2000) assessed the impact of inequality on R&D expenditures for the environment taken "as proxy for the intensity of public engagement in environmental problems" through pooled ordinary least squares and random effects estimations. Using a panel of 19 OECD countries in the period 1980-1991, he finds that higher inequality reduces environmental care, however, the effect is statistically significant at 5% level in the pooled ordinary least squares model only.

Using the principal components analysis, Boyce et al. (1999) statistically estimate a measure of inter-state variations in power distribution based on voter participation, tax fairness, Medicaid accessibility, and educational attainment levels. They find that income inequality, per capita income, race, and ethnicity affect power distribution in the expected directions. Inequality in power distribution is associated with lower environmental policies, and these in

turn are associated with higher environmental stress. Both environmental stress and power inequality are associated with adverse public health outcomes.

Torras and Boyce (1998) examine the effect of income distribution on a set of water and air pollution variables using the Global Environment Monitoring System (GEMS) data, Gini index, adult literacy rates and an aggregate of political rights and civil liberties.

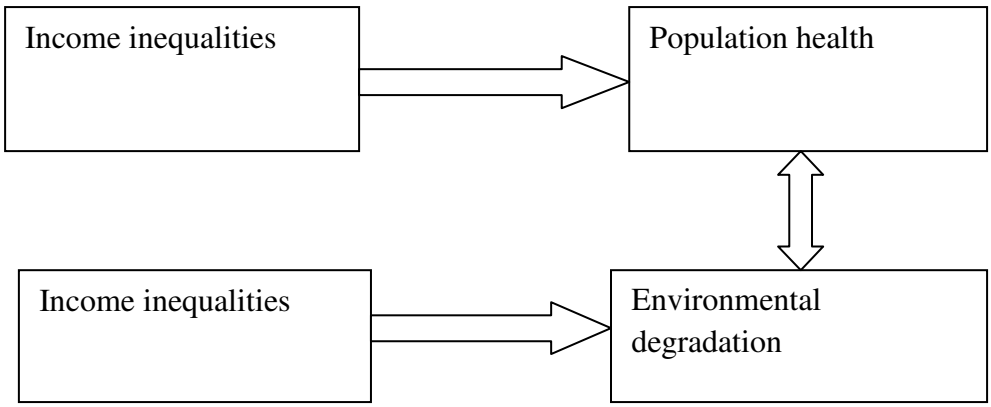
With an OLS estimation, they obtain mixed results on the environmental impact of income inequality. The Gini coefficient is positive for some environmental indicators and negative for others.

It is also possible that more environmental degradation increases income inequality. In fact, environmental degradation in many ways affects the livelihood of the poor. The poorest are vulnerable to environmental degradation since they depend heavily on natural resources and have less alternative resources. They are also exposed to environment hazards and are less capable of coping with environmental risks (Dasgupta & Maler, 1994; World Bank, DFID, EC, UNDP, 2002). Furthermore, the rich are more capable of looking after themselves from environmental diseases than the poorest.[4]

This review explains the complexity of the relation between income distribution and environment. Figure 2.1 summarizes the relation linking income inequality and population's health, by emphasizing what is done in this chapter. Indeed, we combine the literature on the association between income inequality and environment, and that linking environment and income distribution to explore the probably role of pollution as a channel through inequalities affect population health.

[4] This is not the object of the present study.

Figure I.2. 1: Relations between Income level, Income Inequality, Ecological Degradation and Health



Source: Author

2.3. Empirical analysis

2.3.1 Estimations

The analysis is subdivided into three steps. We examine, first, the impact of income inequality on environmental quality. Then, we study the association between environment quality and health status. Finally, we assess these two effects simultaneously.

Based on important existing literature on the determinants of environmental degradation (Heering et al., 2001, Gangadharan and Valenzuela, 2001), the econometric relation between inequality and environment can be written as:

$$\ln(env_{it}) = \lambda_i + \beta INEQ_{it} + \delta_k X_{kit} + \varepsilon_{it} \qquad (2.3.1)$$

Where ln(env) and INEQ represent respectively the logarithm of environment quality and income inequality measure. X_k is the matrix of the control variables. The country fixed effects are represented by λ_i and ε_{it} is the error term.

This equation could be estimated by the Ordinary Least Squares (OLS), but it is very likely that the income distribution variable suffers from endogeneity problem. Indeed, three sources of endogeneity are generally pointed out in the literature. Endogeneity may firstly be caused by the reverse causality between the variable of interest and the dependent variable. Another source of endogeneity is omitted variables bias. This problem occurs when there is a third variable, which could simultaneously affect the variable of interest and the dependent variable. Finally, endogeneity may be caused by measurement error.

The environmental degradation may increase income inequality as explained in section 2, and this potential simultaneity can be a source of endogeneity. In order to solve this problem, we define as instrumental variable the dependency ratio and we estimate equation (2.3.1) with the Two Step Least Square (2SLS) method. As a proxy for demographic variable, age dependency ratio is an important determinant of income inequality because of its distributive effect and it is less convincing to argue that it affects directly environment quality.

In the second model, health status is expressed as a function of environmental quality and other explanatory variables.

$$Health_{it} = \eta_i + \gamma \ln(env_{it}) + \theta_k Z_{kit} + \omega_{it} \qquad (2.3.2)$$

Where *health* represents health status measure and Z_{it} is the matrix of the control variables. η_i represents the country fixed effects and ω_{it} is the error term.

Equation (2.3.2) is estimated with standard fixed effects since we do not expect any potential source of endogeneity of our variable of interest (environment) that may lead to biased estimate of γ. Indeed, in our model, we do not expect any mechanism through which population health may affect environment quality. One could suppose that health may impact environment through its effect on income and development level. Even though this argument

seems less relevant, it cannot affect our identification strategy since we control for development level. To avoid endogeneity problem caused by omitted variables bias in the model, we control for all potential variables which could simultaneously affect the environment quality and population health.

These two equations allow the assessment of the association between income distribution and environment on one hand, and the relation linking health and environment on the other hand. But, it is not sufficient to draw a conclusion whether the health effect of inequality is channelled by environment, since correlation is not transitive. To clearly shed light this effect, we estimate simultaneously equation 2.3.1 and equation 2.3.2.

$$\begin{cases} \ln(env_{it}) = \lambda_i + \beta INEQ_{it} + \delta_k X_{kit} + \varepsilon_{it} \\ Health_{it} = \eta_i + \gamma \ln(env_{it}) + \theta_k Z_{kit} + \omega_{it} \end{cases} \quad (2.3.3)$$

This model is estimated with Three Stages Least Square method (3SLS). It takes into account the likely correlation between the error terms of the two equations, the endogeneity issue of environmental variable, and the heteroscedasticity as well as the serial correlation of the error terms.

2.3.2 Data and variables

The data used in this chapter cover the period 1970-2000 subdivided into 6 periods of 5 years and we retain for the basic regression 90 developed and developing countries (because of data availability, see Appendix 2.2). As health variable we use the logit of under five survival rate (LOGIT SURVIVAL). The under-five survival indicator is limited asymptotically, and an increase in this indicator does not represent the same performance when its initial level is weak or high. The best functional form to examine that is where the variable is expressed into a logistic form, as Grigoriou (2005) underlined, we also use the logarithmic form.

$$\log it\ \text{survival} = \ln\left(\frac{survival}{1-survival}\right).$$

Data on infant and under five mortality rates are from the UN Inter-agency Group (WHO, UNICEF, the World Bank, and UNPD) for Child Mortality Estimation.[5]

The environmental quality is represented by three variables: the particulate matter less than 10 µm aerodynamic diameter (PM10)[6], the biological oxygen demand per capita (BOD) both taken from the World Bank World Development Indicator (WDI 2007) and the sulphur dioxide emission per capita (SO2) from Stern (2005). For these variables, a higher value indicates more environmental degradation. PM10 and SO2 are air pollution indicators and BOD in a water quality indicator.

Income inequality is measured by the Gini coefficient (ranging from 0, low inequality to 1, high inequality) taken from the database created by Galbraith and associates and known as the University of Texas Inequality Project (UTIP) database. It contains two different types of data on inequality: the UTIP-UNIDO and the EHII indexes. The EHII (that we use here) is an index of Estimated Household Income Inequality and is built combining the information in the Deninger and Squire (D&S) data with the information in the UTIP-UNIDO data.[7] The Gini coefficient representes graphically the area between the Lorenz curve and the line of equality.

The other explanatory variables have been chosen from existing published papers (Gangadharan & Valenzuela, 2001). In fact, in the environmental equation, we use:

[5] These data are available at: http://www.childmortality.org/

[6] See Dockery (2009) for a large explanation of particulate air pollution.

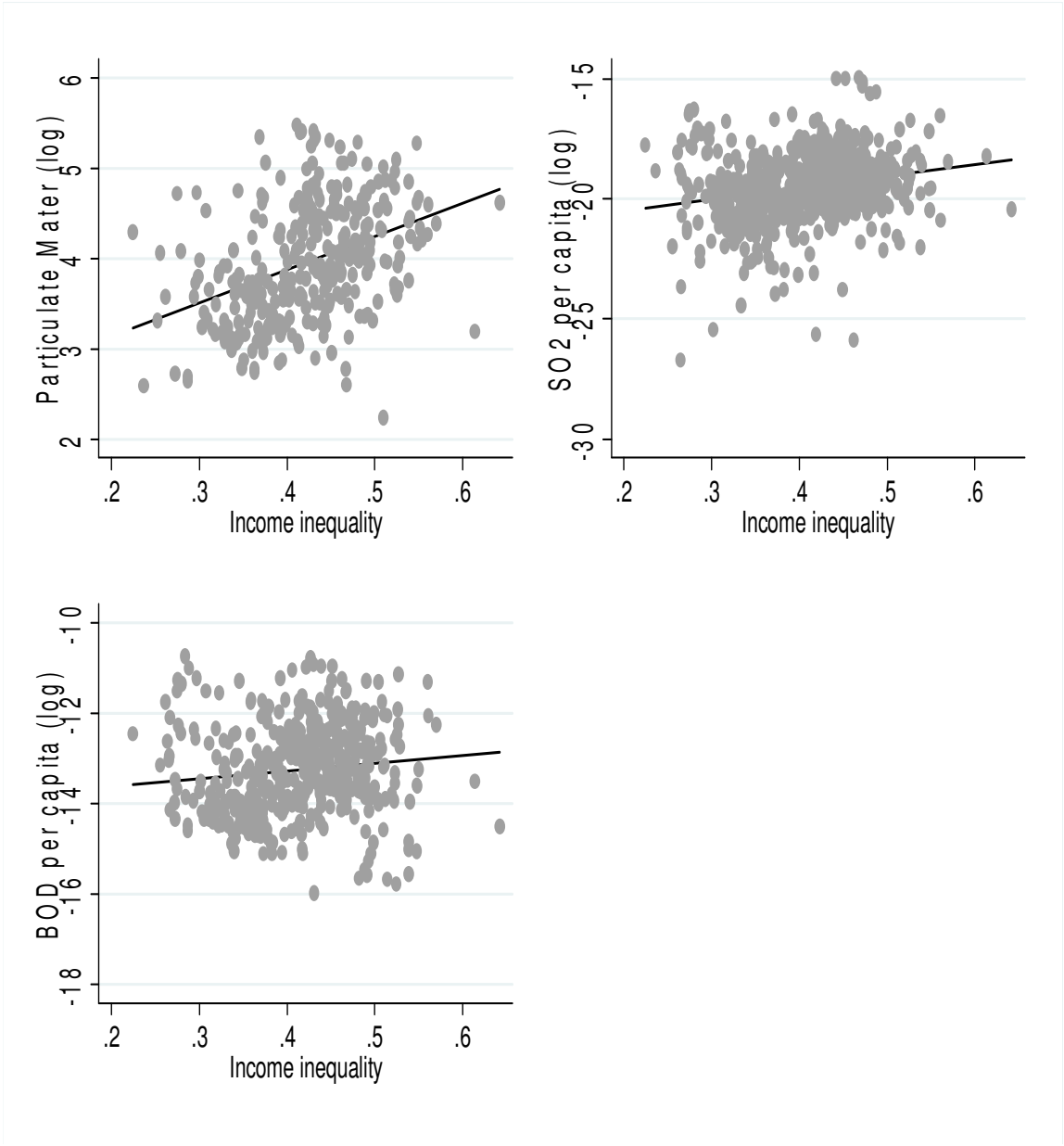
[7] These data are available at: http://utip.gov.utexas.edu/data.html

The gross domestic product per capita (GDPCAP) and its square are introduced to control for the EKC. The hypothesis is verified if the coefficient of GDP per capita is positive and its square negative. We also control for demographic condition via population density (POPDENS) and the percentage of urban population (Urban POP.). Foreign direct investment (FDI), and trade openness (OPEN), are introduced to control for the economic openness of the country. All these indicators as well as the dependency ratio (DEPENDENCY), our instrument of income distribution, are taken from WDI 2007, and the Percentage of "no schooling" in the total population (SCHOOL) from Barro and Lee (2000).

For health equation, we control for the vaccination rate against diphtheria, pertussis and tetanus (DPT), fertility rate (total births per woman) from WDI 2007, and the Percentage of "no schooling" in the total population (SCHOOL).

Appendix 2.3 summarizes the characteristics of the main variables. This appendix shows the mean, the minimum, the maximum, the standard deviation, the coefficient of variation, the characteristics and sources of each variable. These statistics are completed by Appendix 2.4 which presents the correlation between important variables. These statistics are confirmed by Figure 2.2, which displays the statistical relation between inequality and environmental variables. These relations are just a simple correlation and don’t take into account the influence of other variables. The econometric section will solve for this.

Figure I.2. 2: Correlation between income inequality and environment quality



Source: Author

2.3.3 Results

2.3.3.1. Income inequality and environmental quality

Table 2. 1: Impact of income inequality on environment quality

	2SLS FIXED EFFECTS ESTIMATIONS DEPENDENT VARIABLES		
INDEPENDENT VARIABLES	(1) SO2	(2) BOD	(3) PM10
INEQUALITY	1.557*	0.358**	-0.00800
	(1.650)	(2.005)	(-0.0235)
GDPCAP	4.293***	0.0261	-0.681
	(6.623)	(0.193)	(-1.614)
GDPCAP SQUARE	-0.198***	-0.00760	0.0378
	(-4.562)	(-0.859)	(1.393)
POP. DENSITY	1.119***	-0.0224	-0.633***
	(3.093)	(-0.301)	(-2.883)
SCHOOL	-0.188	0.116	-0.388
	(-0.367)	(1.148)	(-1.144)
FDI	-0.308	0.488***	-0.104
	(-0.308)	(2.821)	(-0.310)
OPENNESS	-0.360	-0.198***	0.254***
	(-1.626)	(-5.292)	(2.908)
URBAN POPULATION	2.831***	-0.268*	-0.379
	(3.371)	(-1.664)	(-0.753)
Time dummy	YES	YES	YES
Observations	483	369	214
NB countries	86	87	75
R-squared	0.37	0.21	0.57

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis. Income inequality (INEQUALITY) is instrumented by dependency ratio. The first step estimation results are presented in appendix 2.5.

The results obtained from equation (2.3.1) for the whole sample (developed and developing countries), are reported in Table 2.1. The column 1 of this table shows the results when the logarithm of sulphur dioxide emission per capita (SO2) is used as environmental variable. The environmental Kuznets Curve (EKC) hypothesis is verified, since the coefficient of the logarithm of GDP per capita (GDPCAP) is positive and statistically significant, and the coefficient of its square (GDPCAPSQ) is negative and also significant. In this column, the coefficient of inequality variable (INEQUALITY) is positive and statistically significant at 10%, showing that an increase in income inequality worsens environmental quality.

Columns 2 and 3 summarize the results when the biological demand (BOD) and the particulate matter (PM10) are respectively used as environmental variables. The important results remain unchanged, namely, income inequality is an important cause of environment degradation, except for PM10 where the coefficient of inequality is not statistically significant.

2.3.3.2. Environment and health

The effect of environmental quality on health status (equation 2.3.2) is estimated with standard fixed effects model and the results are reported in table 2.2.

Column 1 presents the results when environment quality is measured by SO2 emission. All the explanatory variables have the expected sign and are statistically significant, except the education indicator (SCHOOL) which is not statistically significant. GDP per capita lagged (GDPCAP) and immunization rate (IMDPT) improve the survival rate while fertility rate (FERT) and environment quality (BOD) degrades it. The negative and significant coefficient of SO2 shows that air pollution worsens health status as expected in the literature review. Columns 2 and 3 show the results when BOD and PM10 are respectively used as

environmental indicators. All these columns underline the negative effect of air and water pollution on population's health.

Table 2. 2: Impact of environment quality on health

	OLS FIXED EFFETS ESTIMATION		
	Dependent variable: Health: (under 5 survival rate)		
INDEPENDENT VARIABLE	1	2	3
GDPCAP	0.548***	0.565***	0.374***
	(10.05)	(8.848)	(4.668)
IMDPT	0.431***	0.515***	0.478***
	(5.418)	(5.505)	(3.918)
SCHOOL	0.108	0.125	0.633
	(0.444)	(0.434)	(1.615)
FERT	-0.208***	-0.185***	-0.123***
	(-7.136)	(-5.395)	(-3.351)
SO2	-0.205***		
	(-8.154)		
BOD		-0.230***	
		(-4.368)	
PM10			-0.436***
			(-5.783)
CONSTANT	-3.554***	-2.539***	1.655**
	(-6.027)	(-3.838)	(2.029)
Time dummy	YES	YES	YES
Observations	432	376	282
R-squared	0.70	0.67	0.59
Number of id	95	93	96

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

2.3.3.3. Income inequality, environment and health

To assess the role of environmental degradation as a channel of transmission of the impact of income inequality on health status, Equation (2.3.1) and (2.3.2) are estimated simultaneously with 3SLS method and the results are presented in Table 2.3.

Table 2. 3: Three stages least square estimation of environmental and health equations

	DEPENDENT VARIABLES:					
INDEPENDENT VARIABLE	(1) SO2	(2) HEALTH	(3) BOD	(4) HEALTH	(5) PM10	(6) HEALTH
INEQUALITY	1.619*		1.514***		3.248***	
	(1.709)		(7.227)		(4.663)	
SO2		-0.217***				
		(-4.363)				
BOD				-0.292		
				(-1.320)		
PM10						-0.370***
						(-4.351)
GDPCAP	2.060***	0.738***	-0.0925	0.559***	1.431***	0.548***
	(5.801)	(14.82)	(-1.196)	(14.57)	(5.407)	(9.918)
GDPCAP SQ	-0.109***		0.00231		-0.0916***	
	(-5.106)		(0.496)		(-5.870)	
POP. DENS.	-0.112***		-0.0589***		0.0521**	
	(-3.254)		(-7.877)		(2.082)	
FDI	-0.846		0.538		-0.139	
	(-0.433)		(1.257)		(-0.117)	
OPENNESS	-0.163		0.0318		-0.296***	
	(-1.130)		(1.025)		(-2.831)	
URBAN POP.	1.516***		-0.0242		0.0940	
	(4.533)		(-0.338)		(0.392)	
SCHOOL	-0.476	-1.250***	-0.235***	-1.094***	1.146***	-0.587**
	(-1.435)	(-7.671)	(-3.519)	(-5.689)	(4.054)	(-2.447)
VACCINATION		0.318**		0.273**		0.427**
		(2.526)		(2.415)		(2.191)
FERTILITY		-0.128***		-0.139***		-0.169***
		(-5.406)		(-4.451)		(-5.076)
CONSTANTE	-21.45***	-5.138***	0	0	-3.218***	0
	(-14.62)	(-5.019)			(-2.887)	
Time dummy	YES	YES	YES	YES	YES	YES
Observations	347	347	344	344	219	219
R-squared	0.54	0.89	0.42	0.91	0.45	0.91

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

The first two columns of this table present the results when environment is measured by SO2 per capita. Columns (3) and (4) show the results when SO2 is replaced by BOD, while the two the results from PM10 as environmental variable are presented in the last two columns. Columns (1), (3) and (5) confirm the results obtained in Table 1, namely increasing income inequality degrades the physical environment. Columns (2), (4) and (6) highlight that, these pollutions from income distribution are harmful for under five mortality rate.

2.4. Conclusion and policy implications

The purpose of this chapter was to investigate the effect of income distribution on health which passes through environmental quality. Theoretically, we show that environment degradation could be consider as a channel through which income inequality affects population health in addition to the traditional mechanisms found in the literature.

Empirically, we demonstrate through an accurate econometric analysis that income inequality affects negatively environmental quality and this environmental degradation worsens population's health. This confirms that environment quality is an important channel through which income inequality affects population health. These results hold for air pollution indicators (PM10 and SO2) and water pollution indicator (BOD). It is also robust for rich and developing countries.

As policy implication, our results mean that income inequality is bad for health and environment, and countries with high income inequality may implement distributive policy in order to avoid its negative impact on health. Moreover, this chapter underlines the importance of income distribution in the achievement of the Millennium Development Goals.

International community as well as governments should pay more attention to the consequences of their policies on income inequality in order to improve health outcomes and physical environment quality.

The allocation of resources either in the form of public programs or direct public investment in health and environmental infrastructure, should focus on targeting the income gaps in the communities rather than poor households only, Because investments in the reduction of inequalities have an externality effect on household health and environment. Publicly funded programs need to recognize and capture this externality.

Given the importance of our findings for policy makers, they should be confirmed or extended by future researches. This work is based on country level data. One way it could be extended is by exploring individual or state level data in order to confirm or reject our results. We have just used three environmental indicators and Gini coefficient as income inequality indicator. Another way to extend it is to verify whether our conclusions are robust or not to other environmental and inequality variables.

APPENDICES 2

Appendix 2.1: literature review on the empirical studies linking income inequality and environment.

study	year	inequality variable	environment measure	effect of inequality		data	estimator	review	other covariates
				effect	sig. Level				
Clément and Meunie	2008	gini WIDER	SO2 emission	impr.	10%	83 developing and transition countries in 1988-2003	OLS	Cahiers du GREThA n° 2008-13	GDP, GDP², GDP [3]
			BOD emission	degr.	1%				
Heering, N., Mulatu A. and, Bulte E.	2001	gini index	access to safe water, access to sanitation, and deforestation	degr.	1%	16-country sample of sub-Saharan African countries	pooled	Ecological Economics 38, 359–367	GDP, GDP²
			carbon dioxide emissions, nitrogen depletion, and phosphorus depletion	impr.	1%				
			sulfur dioxide	impr.	NO				

study	year	inequality variable	environment measure	effect of inequality		data	estimator	review	other covariates
				effect	sig. Level				
			and particulate concentrations						
Borghesi	2000	Gini (Deninger and Squire)	CO2 per capita	impr.	1%	panel of 37 countries from 1988-1995	OLS pooled model	NOTA DI LAVORO 83.2000	GDP, GDP², GDP³, Population density, industry share.
				degr.	NO		fixed effects		
Marsiliani and Renström	2000	ratio of households ranked at top 90th percentile to the median household	sulfur, Nitrogen oxides and carbon dioxide	degr.	1%	two panels of 7 and 10 industrialized countries over 1978-1997	simple OLS	CentER working paper n.2000-34	GDP
							ML		
				impr.			fixed effects		
Magnani	2000	quintiles 1 / quintiles 4	Public R&D expenditure for environmental protection	degr.	10%	17 developed countries	fixed effects & random effects	Ecological Economics 32 (2000) 440 431–443	GDP, GDP², Time trend
		gini			NO				
Ravallion M., Heil	2000	gini index	CO2 per capita	impr.	5%	panel of 42 countries in	fixed effects &	Oxford Econom	GDP, GDP²,

study	year	inequality variable	environment measure	effect of inequality		data	estimator	review	other covariates
				effect	sig. Level				
M., Jalan			emission			the period 1975-92	pooled OLS	ic Papers, *52*:651-669	Population
Boyce et al.	1999	power inequality	environment policy	degr.	1%	50 US states in 1990's	OLS	Ecological Economics 29 (1999) 127–140	manufacturing share, urbanization and population density
Scruggs L.A.	1998	Gini (Deninger and Squire)	sulfur dioxide	impr.	1%	25–29 countries for 3 periods: 1979–1982, 1983–1986 and 1987–1990	OLS pooled model	Ecological Economics 26 (1998) 259–275	Democracy, Income, Industrialize site, periode
			particulate matter	impr.	NO				
			fecal coliform	degr.	NO				
			dissolved oxygen	degr.	1%				
Torras and Boyce	1998	gini (low income)	Sulfur dioxide	degr.	1%	287 stations in 58 countries	OLS	Ecological Economics 25 (1998) 147–160	GDP, GDP^2, GDP^3, literacy rate, right
			Smoke	degr.	1%				
			Heavy particles	impr.	1%				
			Dissolved oxygen	impr.	1%				

study	year	inequality variable	environment measure	effect of inequality		data	estimator	review	other covariates
				effect	sig. Level				
			Fecal coliform	impr.	NO				
			Safe water (%)	degr.	1%				
			Sanitation (%)	degr.	NO				
		gini (high income)	Sulfur dioxide	impr.	1%				
			Smoke	impr.	NO				
			Heavy particles	degr.	NO				
			Dissolved oxygen	degr.	NO				
			Fecal coliform	impr.	1%				
			Safe water (%)	degr.	NO				
			Sanitation (%)	degr.	NO				

Appendix 2.2: Country list

World bank	country		World bank	country
ARG	Argentina		JOR	Jordan
AUS	Australia		JPN	Japan
AUT	Austria		KEN	Kenya
BDI	Burundi		KOR	Korea, Rep.
BEL	Belgium		KWT	Kuwait
BEN	Benin		LBR	Liberia
BGD	Bangladesh		LKA	Sri Lanka
BOL	Bolivia		LSO	Lesotho
BRA	Brazil		MEX	Mexico
BWA	Botswana		MOZ	Mozambique
CAF	Central African Republic		MUS	Mauritius
CAN	Canada		MWI	Malawi
CHL	Chile		MYS	Malaysia
CHN	China		NIC	Nicaragua
CMR	Cameroon		NLD	Netherlands
COG	Congo, Rep.		NOR	Norway
COL	Colombia		NPL	Nepal
CRI	Costa Rica		NZL	New Zealand
CYP	Cyprus		PAK	Pakistan
DEU	Germany		PAN	Panama
DNK	Denmark		PER	Peru
DOM	Dominican Republic		PHL	Philippines
DZA	Algeria		PNG	Papua New Guinea
ECU	Ecuador		POL	Poland
EGY	Egypt, Arab Rep.		PRT	Portugal
ESP	Spain		PRY	Paraguay
FIN	Finland		RWA	Rwanda
FJI	Fiji		SEN	Senegal
FRA	France		SLE	Sierra Leone
GBR	United Kingdom		SLV	El Salvador
GHA	Ghana		SWE	Sweden
GMB	Gambia, The		SWZ	Swaziland
GRC	Greece		SYR	Syrian Arab Republic
GTM	Guatemala		TGO	Togo
HND	Honduras		THA	Thailand
HTI	Haiti		TTO	Trinidad and Tobago
HUN	Hungary		TUN	Tunisia
IDN	Indonesia		TUR	Turkey
IND	India		UGA	Uganda
IRL	Ireland		URY	Uruguay
IRN	Iran, Islamic Rep.		USA	United States
ISL	Iceland		VEN	Venezuela, RB
ISR	Israel		ZAF	South Africa
ITA	Italy		ZMB	Zambia
JAM	Jamaica		ZWE	Zimbabwe

Appendix 2.3: descriptive statistics

	MEAN	MINIMUM	MAXIMUM	COEF. VAR.	STAND. DEV.	NB. OBS.	CHARACTERISTICS	SOURCES
LOGIT SURVIVAL	2,988	0,672	5,293	0,4062438	1,214262	478	logit of survival rate (log survival/log(1-survival))	WHO
PM10	65,858	13,410	237	0,7147875	47,07434	224	carbon dioxide emission as ratio of GDP	WDI 2007
BOD	0,198	0,116	0,342	0,2399657	0,0474592	220	biological oxygen demand as ratio of GDP	WDI 2007
SO2	0,000	0,000	0,001	2,688387	0,0000455	223	sulfur dioxide emission as ratio of GDP	Stern 2004
INEQUALITY	0,417	0,266	0,642	0,1473903	0,0615115	485	Estimated Household Income Inequality	UTIP database
GDPCAP	6280	122,617	36160	1,261498	7922,295	485	Gross Domestic Product per capita	WDI 2007
SCHOOL	0,305	0,000	0,930	0,8898199	0,271307	485	Unschooled population	WDI 2007
IMDPT	0,711	0,012	0,990	0,3504164	0,2490928	351	Immunization rate	WDI 2007
FERT	3,997	1,180	8,494	0,4924775	1,968499	485	fertility rate	WDI 2007
POPDENS	98,714	1,568	951,972	1,26521	124,894	485	population density	WDI 2007
URBAN POP.	0,560	0,053	0,982	0,4255331	0,2382587	224	Proportion of urban population	WDI 2007

Appendix 2.4: correlations between important variables

	LOGIT SURVIVAL	CO2	BOD	SO2	EHII	GDPCAP	SCHOOL	IMDPT	FERT	POPDENS
LOGIT SURVIVAL	0.94*									
LIFE EXPECT	0.30*	1.00								
CO2	-0.45*	0.01	1.00							
BOD	-0.19*	0.06	0.20*	1.00						
SO2	-0.62*	-0.17*	0.13*	0.11*	1.00					
EHII	0.81*	0.17*	-0.47*	-0.14*	-0.61*	1.00				
GDPCAP	-0.86*	-0.29*	0.33*	0.12*	0.52*	-0.63*	1.00			
SCHOOL	0.64*	0.17*	-0.20*	-0.03*	-0.30*	0.44*	-0.59*	1.00		
FERT	-0.90*	-0.30*	0.32*	0.22*	0.57*	-0.68*	0.84*	-0.61*	1.00	
POPDENS	0.17*	-0.01	0.12*	-0.11*	-0.11*	0.11*	-0.12*	0.05	-0.25*	1.00
FERTILIZER	0.40*	0.02	-0.11*	-0.08*	-0.27*	0.41*	-0.31*	0.25*	-0.32*	0.12*

*significant at 10%.

Appendix 2.6: First step estimation results

	DEPENDENT VARIABLES (FIRST STEP ESTIMATIONS)
	(1)
INDEPENDENT VARIABLES	INEQUALITY
GDPCAP	-0,146
	-2,99
GDPCAPSQ	0,007
	2,63
POPDENS	0,047
	3,46
SCHOOL	0,0023
	0,08
URBAN POP.	-8,14E-06
	-2,48
FDI	0,0635
	0,88
OPEN	0,0036
	0,25
DEPENDENCY	-0,0031
	-3,24
Observations	367
NB countries	86

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

Chapter 3: Intra Countries Health Inequalities and Air Pollution in Developing Countries: Do Political Institutions protect the poor?

3.1. Introduction

The importance of human capital in general and population health in particular as a component of economic development predictors, has been investigated by many scholars (Cuddington & Hancock, 1994; Caselli et al., 1996; Bhargava et al., 2001; Carstensen & Gundlach, 2006; Sachs & Warner, 1997). It is recognized by economists as well as international community that health contributes largely to the improvement of population welfare and economic growth through productivity and availability of healthy workforce (Bloom et al., 2001; Weil, 2007). Environment quality is commonly accepted as one of these determinants that may influence population health. Indeed, many studies have assessed the association between air pollution and health status through macroeconomic studies (Gangadharan & Valenzuela, 2001) as well as microeconomic studies (Burnett & Krewski, 1994; Jerrett et al., 2005). Some authors showed that air quality degradation increases all causes mortality (Woodruff et al., 1997; Gangadharan & Valenzuela, 2001; Chay et al. 2003; Aunan & Pan, 2004; Jerrett et al., 2005) while others confirm its impact on cause-specific mortality or morbidity (Aunan & Pan, 2004; Burnett & Krewski, 1994; Jerrett et al., 2005). Moreover, other scholars investigated the heterogeneity in the health effect of air pollution according to socioeconomic status (Charafeddine & Boden, 2008; O'Neill et al., 2003), but these studies remain theoretical or specific in a given region and focus only on health status. In addition, international studies on this topic are based on average health in the population. One of the drawbacks of the use of average health is its inability to take into account the extent of health disparities within a population, given the differential in policy response.[8] This can be solved by using health distribution. In this chapter, we investigate how air pollution

[8] Sahn et al. (2003) demonstrate within-country variation is the source of most inequality, rather than the differences between countries

may impact income related health inequality within a country and the role of political institutions in such relation using data from developing countries.

Some theoretical arguments - namely, heterogeneity in exposition to air pollution among income classes, prevention ability against health effect of environment degradation, ability to respond to sickness caused by pollutants and susceptibility of some groups to air pollution effect – allow us to predict a larger impact of pollution on the poorest as compare to its effect on the richest class of income. Therefore, this may increase income related health disparities among the population. Good political institutions may mitigate this health inequality effect of environmental degradation through universal health policy issues, information and advices about health practices, and health infrastructures availability.

This chapter is different from previous literature since it is the first, from our knowledge, that explicitly links air pollution to within country health inequalities. Moreover, it uses a rich database from the World Bank that allows us to take into account both within and between countries characteristics of health outcomes.

Our empirical results confirm our theoretical expectations. Indeed, air pollution degrades population health and the poorest populations suffer more from this degradation than the richest. This heterogeneity in health consequences of pollution is alleviated by good political institutions.

The rest of the chapter is organized as follows. In section 2 we define and discuss the different measures of health inequalities in the literature. Section 3 develops the theoretical links between health inequalities, air pollution and political environment. In this section we explore how environmental degradation may increase this disparities and the role of institutions quality. Section 4 is devoted to the empirical design. We expose the econometric methodology and the data we use in this section. The results are presented in section 5 and section 6 presents some robustness checks. Finally section 7 concludes.

3.2. Health inequality: definition and measures

Health inequality in a population can be defined as the differences, variations, and disparities in health achievements among individuals or groups of this population. This descriptive term includes health inequity which is the normative part of health inequality since it depends on personal judgement (Kawachi et al. 2002; Braveman and Tarimo, 2002).[9] As argued by Deaton & Paxson (1998), the measurement of health inequality raises at least two important issues. First, the identification of a reliable and available measure of health status data can be considered as a challenge. Several indicators are suggested in the literature, but all of them are source of critics or suffer from data unavailability. Fang et al. (2010) classified these indicators into two categories. The traditional one based on ill health incidents such as vital statistics, disease statistics and children growth data. The second category constituted of newer indicators focuses on healthy life span such as potential years of life lost (PYLL), life expectancy free of disability (LEFD), active life expectancy (ALE), disability adjusted life years (DALY) and disability adjusted life expectancy (DALE). Another important issue is whether the chosen indicator is qualitative or quantitative. The qualitative or categorical data prevents the straightforward use of traditional tools of distributional analysis, such as the Lorenz curve, in evaluating inequality. Allison & Foster (2004) present a methodology for evaluating overall inequality in health when the data are qualitative rather than quantitative in nature.

Once the appropriated measure of health is identified, the second issue is how to measure inequality in health status. In economic literature, health inequality is assessed through two different approaches. On the one hand, some scholars measure health inequality through the

[9] Some determinants of intra country health inequality are presented in appendix 3.1.

distribution of health status across individuals in a population, like measures of income distribution in a population (Legrand 1987; Kawachi et al. 2002; Sahn et al., 2003, Sahn, 2009). Indicators from this approach include the Lorenz curve, the gini coefficient or other measures of health dispersion (Wagstaff & van Doorslaer, 2004). On the other hand researchers assess health distribution by measuring health difference across social groups (income class, social class, age, race, place or neighbouring) and these indicators include the index of dissimilarity (ID), the slope relative indices of inequality, the index of concentration, the range, the pseudo lorenz curve, the adapted gini coefficient. Some measures that are based on both health and social position utilize the ordered nature of socioeconomic status (the slope and the concentration index) while others including the adapted Gini coefficient and the index of dissimilarity do not.

As argue by Kunst (2008), the choice of measuring method depends on the health outcomes of interest, the data sources that can be accessed, and the socioeconomic information that is available. For Manor et al. (1997), the measures based only on the distribution of health are inadequate in examining social inequalities in health. The joint distribution of both health and socioeconomic status should be considered in this context. Wagstaff et al. (1991) and Schneider et al. (2002) detailed the calculation methods and the advantages and disadvantages of the various measurements. According to Szwarcwald (2002), the measure of variations in health status across individuals in a population depends at the same time on the performance of the health system in diminishing the socioeconomic health inequalities and the extent of the income inequalities in the population. So, it is a matter of choice whether one should or should not consider the distribution of the population across socioeconomic groups. If one considers that what is important about health inequalities is to assess the magnitude of the inter-individual differences in health status, the index of health inequalities will inevitably reflect the inequality in socioeconomic status. If the main goal is to assess the performance of

health systems, this is clearly a restriction because the extent of inequalities in socioeconomic status within the population is generally outside the field of control of public health policies and actions. According to Levine et al. (2001) inequality in health is a relative rather than an absolute concept, and ratios rather than absolute differences are a more valid measure of inequality. They calculated time series for black/white ratios of age-adjusted, all-cause mortality and life expectancy in the USA. Lai et al. (2008) used two classes of generalized Gini coefficients (G1 and G2) of life expectancy to measure health inequalities among the provinces of China and the states of the United States. G1 is the measure of individual/mean absolute differences and G2 measures inter-individual absolute differences. For China, their results indicated that there was statistically significant health inequality by both G1 and G2. However, for the US, their results showed that there was significant health inequality by G1 but no statistical significance was found in health inequality by G2. Overall, from their study, China has higher health inequality than the United States.

In this chapter, the second approach of measuring health inequality is used. More precisely, we compare health status between income quintile classes.

3.3. Health inequality, pollution and institutions quality

A healthy labour force is essential for the development of an economy and requires a healthy environment (clean air, water, recreation and wilderness). As argue by Pearce & Warford (1993), the immediate and most important consequences of environmental degradation are damage to human health through different forms of diseases. Many authors have assessed how air quality may be associated to population's health. Scholars showed that air pollution may increase mortality rate (Woodruff et al., 1997; Gangadharan & Valenzuela, 2001; Chay et al. 2003; Aunan & Pan, 2004; Jerrett et al., 2005). Aunan & Pan (2004) propose exposure-response functions for health effects of PM_{10} and SO_2 pollution in China, based on Chinese

epidemiological studies. They found 0.03% (S.E. 0.01) and 0.04% (S.E. 0.01) increase in all-cause mortality per $\mu g/m^3$ PM_{10} and SO_2, respectively. Furthermore, Jerrett et al. (2005) investigated whether chronic exposure to particulate air pollution is significantly associated with mortality when the effects of other social, demographic, and lifestyle confounders are taken into account. Their results show substantively large and statistically significant health effects for women and men.

The link between pollution and particular illness, such as cardio-respiratory disease (Aunan & Pan, 2004; Burnett & Krewski, 1994; Jerrett et al., 2005), asthma (Nauenberg & Basu, 1999) and congenital anomalies (Rankin et al., 2009) was also studied. Burnett & Krewski (1994) find strong associations between the number of daily health events (hospital admissions or emergency-room visits for respiratory illnesses) and daily levels of ambient air pollutants in the vicinity of several hospitals with data obtained from 164 acute-care hospitals in Ontario over the May-to-August period from 1983 to 1988 and a random-effects relative-risk regression model. Rankin et al. (2009) investigate the association between exposure to particulate matter with aerodynamic diameter less than 4 mg/m^3 (BS) and sulphur dioxide (SO_2) during the first trimester of pregnancy and risk of congenital anomalies through a case–control study design among deliveries to mothers resident in the UK Northern health region during 1985–1990 and logistic regression models. They found a significant but weak positive association between nervous system anomalies and BS, but not with other anomaly subtypes. For SO2, they found a significant negative association with congenital heart disease combined and patent ductus arteriosus.

In addition to the effect of air pollution on population health, this chapter assesses the association between pollution and income related health inequalities within a country. At least three theoretical arguments allow the expectation of a positive association between physical environment quality and inequalities in health. Firstly, air pollution exposure is differentially

distributed by income level. Indeed, poor communities are more likely to be exposed than others, since they generally live in more polluted area and they cannot afford moving from polluted area to a less polluted one. That is at the core of environment justice movement. Moreover, poor people are more exposed to pollutants at work. Populations with less wealth are more likely to be employed in dirtier occupations and may also be more likely to be exposed to pollutants indoors from heating and cooking. That may be due to the low and less prestigious position their generally occupied. The heterogeneity of exposure over space varies by pollutant type. Fine particles are distributed fairly homogeneously over large urban areas due mostly to the contribution of small, long-range transport particles (O'Neill et al., 2003). Secondly, at a given level of exposition, rich communities have more prevention than poor. In fact, because their parents are poor, some children do not have access to immunization against illness caused or conveyed by air pollution such as meningitis. Poor communities may also lack access to stores that sell fresh fruits and vegetables or the income to buy them, resulting in reduced intake of antioxidant vitamins that can protect against adverse consequences of air pollution exposure (Romieu et al., 1998; O'Neill et al., 2003). Another way of prevention is to respect certain rules of hygiene. For example, protection of foods by covering them and the purchase of packaged products may reduce the health consequences of exposure. But these rules are more respected by the rich than the poor because of education and financial reasons. This differential prevention deepens inequalities in health caused by pollution since it mitigates the consequences for the wealthier. Finally, differential access to medical care (because of inequalities in access to health insurance) is another fact explaining inequalities in the health effect of air pollution. Indeed, poor people may not have the appropriate prescription for a respiratory condition such as asthma. Medication can alleviate symptoms aggravated by pollution exposure, and more consistent use of corticosteroids lowers baseline inflammation, potentially lowering responsiveness to pro-inflammatory pollutants (O'Neill et

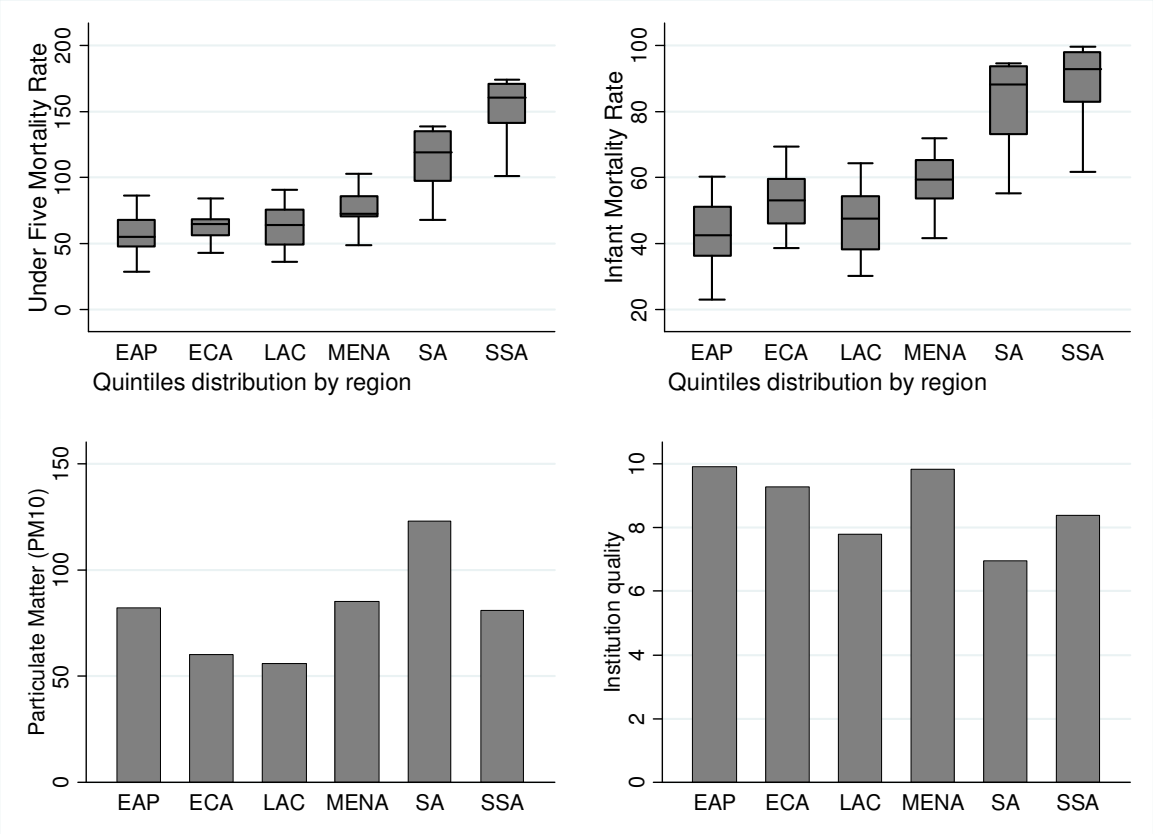
al., 2003). All that arguments increase the vulnerability of income disadvantaged population as compare to the richest. Makri & Stilianakis (2008) identify and evaluate information on population characteristics associated with vulnerability to ambient air pollution from a risk analysis perspective and based on available evidence. They found higher risks for foetuses and children, the elderly, and persons with pre-existing diseases. They also found that epidemiologic evidence of higher risks for racial minorities and social economically disadvantaged populations may be partly related to physiological capacity due to pre-existing diseases as well as health status. Charafeddine & Boden (2008) showed that income inequality plays a modifier role in the association between general self-reported health and particulate pollution. They hypothesize that individuals living in states with lower income inequality are significantly more likely to report fair or poor health if they lived in counties where particulate pollution is high. But, their results contradict their hypothesis.

In countries with good institutions, these disparities in health effect of air pollution could be mitigated. Institutions are understood here as democratic principles, such as regular elections, universal suffrage, representation, one person–one vote, multiparty competition, and civil liberties. Thus, good institutions might produce competition for popular support among leaders who are trying to conserve or win elected office. Democratic institutions might therefore reduce health effect of pollution of the poor through their general impact on universal health policy issues, such as universal access to high quality services and universal health insurance and accessible programs. Good institutions may in addition, provide information and advices about hygiene, good health practice, and other knowledge useful for the population in general, and the poorest in particular. Political institutions could also alleviate social disparities and income inequalities that results from greater political voice and participation. Finally, governments are likely to build infrastructures (road, hospital) that could reduce air pollution or its effect for the poor. By contrast, authoritarian regimes prevent

human development, since its improvement mobilizes citizens to advocate for greater participation and more resources (Ruger, 2005).

Figure 3.1 depicts the inter quintiles distribution of mortality rates among regions (top graphs) as well as pollution level (bottom left) and institutional quality (bottom right). From this figure we can notice that mortality rates are more unequally distributed in Sub Saharan Africa (SSA) and South Asia (SA) than other region.

Figure I.3. 1: Distributions of Mortality rates and its link to pollution and institution by region



Source: Construction of author

These regions are also those with more Particulate Matter (PM10) emission. Middle East and North Africa (MENA) and East Asia and Pacific (EAP) also experience high pollution level, but inter quintiles health inequality is not very large. This may be due to the fact they have the

best political institutions. This statistically shows that there is a link between health inequality, air pollution and political institutions.

3.4. Empirical design

3.4.1 Estimation methodology

The object of this chapter is to evaluate the effect of air pollution on income related health inequalities and the role of political institutions in mitigating such impact. For this purpose, three econometric models are successively estimated:

The first equation assesses the effect of air pollution on health inequality between countries, while controlling for other potential determinants of health outcomes. Based on some existing empirical works (Gwatkin et al., 2007; Berthelemy & Seban, 2009; white et al., 2003), the following model is specified:

$$health_{ijt} = X'_{ijt}\beta + \delta environment_{jt} + \mu_i + \varepsilon_{ijt} \qquad (3.4.1)$$

Where, the variable $health_{ijt}$ represents the health outcomes (infant and child mortality rates) of the i^{th} quintile in country *j* in the year *t*. *environment* represents the variable of air pollution (sulphur dioxide emission per capita and particulate matter) and *X* is the vector of control variables (mother education, gross domestic product per capita, immunization rate against DPT, fertility rate, population density and the percentage of urban population). μ_i represents the quintile fixed effect and ε_{ijt} is the error terms. In this model, the coefficient of the environmental variable (δ) is of special interest. We expect a positive coefficient since this expresses the deterioration of population health caused by an increasing in environment pollution (marginal effect).

This equation is estimated with the ordinary least squares since we do not expect any potential source of endogeneity (reverse causality, omitted variables, measurement errors) of our variable of interest (environment) that may lead to biased estimate of δ.

In order to assess the heterogeneity in the effect of pollution on health within population, we add the interactions terms of quintile dummies and environmental variables and we obtain the following model:

$$health_{ijt} = X_{ijt}^{'}\beta + \delta environment_{jt} + \sum_{i=2}^{5} \lambda_i (environment_{jt} * \mu_i) + \mu_i + \varepsilon_{ijt} \qquad (3.4.2)$$

In this model the marginal effect of air pollution on quintile i^{th}'s health outcomes is: $\frac{\partial(health_i)}{\partial(environment)} = \delta + \lambda_i$. We expect a higher impact of environment degradation on health for poor income quintile as compare to richer ones ($\lambda_2 > \lambda_3 > \lambda_4 > \lambda_5$) and environment quality may be considered as a determinant of income related health inequality. Similar approach is used in Fay et al. (2005), Berthelemy & Seban (2009).

Finally, we assess whether political institutions may mitigate this gap in the health effect of environment among poor and rich income classes. For this aim, we include in equation (3.4.2) the interaction term of environment, quintile dummies and institution variable, the interaction term of environment and institution variable, and the interaction term of institution and quintile dummies. The third model can be written as follows:

$$\begin{aligned} health_{ijt} = {} & X_{ijt}^{'}\beta + \delta environment_{jt} + \sum_{i=2}^{5} \lambda_i (environment_{jt} * \mu_i) + \sum_{i=2}^{5} \varphi_i (institution_{jt} * \mu_i) \\ & + \psi (environment_{jt} * institution) + \sum_{i=2}^{5} \gamma_i (environment_{jt} * institution * \mu_i) + \mu_i + \varepsilon_{ijt} \end{aligned} \qquad (3.4.3)$$

Where, *institution* denotes political institution variables. The marginal effect of environment on the health outcomes of quintile i becomes: $\frac{\partial(health_i)}{\partial(environment)} = \delta + \lambda_i + (\psi + \gamma_i) * institution$.

This marginal effect depends on institutions quality, and its effect is given by: $\frac{\partial^2(health_i)}{\partial(environment)\partial(institution)} = \psi + \gamma_i$. Political institutions alleviate the disparities in the health effect of environment if γ is higher for rich income classes as compare to poor income quintiles, namely, $\gamma_2 < \gamma_3 < \gamma_4 < \gamma_5$.

Like the first equation (3.4.1), equations (3.4.2) and (3.4.3) are estimated with ordinary least squares and we make a cluster for each country, and all variables are expressed in natural logarithm.

3.4.2 Data and variables

Data come from different sources and are largely utilized in health economics literature.

Health outcomes: Data on health variables are taken from the study leaded by Gwatkin *and al.* (2007) on Health, Nutrition and Population in 56 developing countries (see Appendix 3.2), and all the data are disaggregated by income quintiles. In this database, more than half of the countries are African. The report of Gwatkin *et al.* (2007) is based on data drawn from several demographic and health surveys (DHS) conducted in these countries. These surveys target especially maternal and child health with a standardized questionnaire. Data also include socioeconomic variables like mother education for each quintile.

The report includes several indicators of health status and utilization of health services. In this chapter, we are only interested in infant and under five mortality rates. These data have already been used in the literature by Fay et al. (2005), Ravallion (2007), McGillivray et al. (2008) and Berthelemy & Seban (2009). We use the logistic form of mortality rates.[10]

[10] The mortality indicators are limited asymptotically, and an increase in this indicator does not represent the same performance when its initial level is weak or high, the best functional form to examine is that where the variable is expressed as a logit (Grigoriou 2005).

Appendix 3.3 presents important statistics of health, education and fertility indicators for each income quintile. This table points out the large disparities among income classes in favor of rich people for all these variables. Figure 3.2 confirms this inequality for mortality rates.

Environmental quality variable: Air pollution is represented by two indicators. The first is sulphur dioxide emission per capita (SO_2) taken from the database compiled by stern (2005) and used in many papers (De Melo et al., 2008). The second environmental indicator is particulate matter less than 10 µm aerodynamic diameter (PM10)[11] taken from World Development Indicator 2007 (WDI 2007).

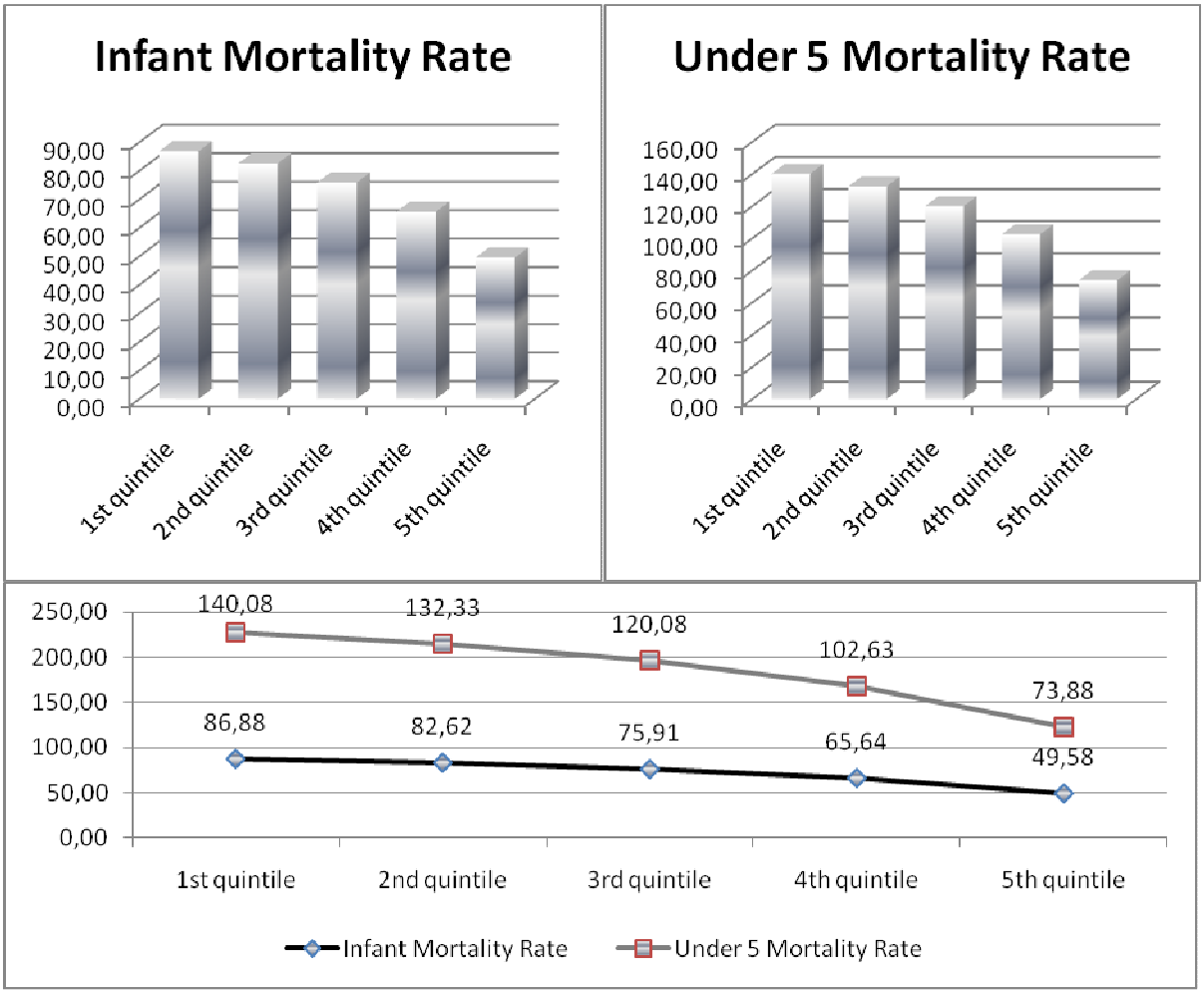
Institution indicators: There are many sources of institution data. Here, we used indicators compiled by "International Country Risk Guide" (ICRG) and freedom house (corruption, military in politics, bureaucracy quality, law and order, democracy accountability and internal conflict indices for ICRG and freedom status index for freedom house). The ICRG model for forecasting political risk was created in 1980 by the editors of International Reports, a weekly newsletter on international finance and economics. They produce a comprehensive system that enables various types of risk to be measured and compared between countries. The system is based on a set of components for political risk. Each component is assigned a maximum numerical value (risk points), with the highest number of points indicating the lowest potential risk for that component and the lowest number (0) indicating the highest potential risk.

Government Stability index is an assessment both of the government's ability to carry out its declared program(s), and its ability to stay in office. The risk rating assigned is the sum of

[11] See Dockery (2009) for a large explanation of particulate air pollution.

three subcomponents (Government Unity, Legislative Strength and Popular Support). Corruption index is an assessment of corruption within the political system.

Figure I.3. 2: Comparison of mortality rates among asset quintiles



Source: Author's construction with data from Gwatkin et al. (2007)

Such corruption is a threat to foreign investment for several reasons: it distorts the economic and financial environment; it reduces the efficiency of government and business by enabling people to assume positions of power through patronage rather than ability; and, last but not least, introduces an inherent instability into the political process.

The institutional strength and quality of the bureaucracy is another shock absorber that tends to minimize revisions of policy when governments change. Therefore, high points are given to countries where the bureaucracy has the strength and expertise to govern without drastic changes in policy or interruptions in government services. In these low-risk countries, the bureaucracy tends to be somewhat autonomous from political pressure and to have an established mechanism for recruitment and training. Countries that lack the cushioning effect of a strong bureaucracy receive low points because a change in government tends to be traumatic in terms of policy formulation and day-to-day administrative functions.

The military is not elected by anyone. Therefore, its involvement in politics is a diminution of democratic accountability. However, it also has other significant implications.

Democracy Accountability is a measure of how responsive government is to its people, on the basis that the less responsive it is, the more likely it is that the government will fall, peacefully in a democratic society, but possibly violently in a non-democratic one. The points in this component are awarded on the basis of the type of governance enjoyed by the country in question.

Law and Order are assessed separately. The Law sub-component is an assessment of the strength and impartiality of the legal system, while the Order sub-component is an assessment of popular observance of the law.

Internal Conflict is an assessment of political violence in the country and its actual or potential impact on governance. The highest rating is given to those countries where there is no armed or civil opposition to the government and the government does not indulge in arbitrary violence, direct or indirect, against its own people. The lowest rating is given to a country embroiled in an on-going civil war. The risk rating assigned is the sum of three subcomponents (Civil War/Coup Threat, Terrorism/Political Violence and Civil Disorder).

Other explanatory variables: As variables of control, we use several indicators. Schooling in the population is represented by mother education. Data about this indicator are taken from Gwatkin et al. (2007). We also control for Gross Domestic Product (GDP) per capita, immunization rate against DPT, fertility rate, population density and the percentage of urban population, all taken from WDI (2007). Finally, year and quintile fixed effects dummies are used and we make a cluster for each country, given data availability. Appendix 3.3 displays the characteristics of health and education data for each quintile while Appendix 3.4 summarizes the characteristics, and sources of each indicator used in this chapter.

3.5. Results

3.5.1 Impact of air pollution on inter countries health inequality

In this subsection, we access the effect of air pollution on health inequality between countries. More precisely, this part presents the results obtained from the estimation of equation (3.4.1). These results are summarized in Table 3.1, with logit of infant and under five mortality rates as dependent variables, and sulphur dioxide and particulate emissions as environmental variables. Regarding the impact of our variables of interest, we find that the elasticity of infant and child mortality rates with respect to environmental variables is positive and statistically significant for each health outcome and each pollution variable. These coefficients indicate that environmental degradation worsens population health outcomes and explains in part health inequalities between countries. These results are in conformity with the literature on this topic as well as our theoretical hypothesis. Our important variables of control also present the expected signs and are statistically significant. Indeed, increasing in Gross Domestic Product per capita (GDP), mother education and immunization rate improve significantly health outcomes while fertility rate degrades them.

Table 3. 1: Impact of air pollution on health inequalities between countries

	Dependent variables			
	(1)	(2)	(3)	(4)
independent variables	child mortality	infant mortality	child mortality	infant mortality
Sulphur dioxide emission (SO2)	0.0861**	0.0695**		
	(2.610)	(2.692)		
Particulate Matter (PM10)			0.125**	0.127**
			(2.092)	(2.254)
fertility rate	0.521***	0.342***	0.627***	0.451***
	(5.000)	(3.233)	(5.125)	(3.530)
schooling	-0.0661	-0.0616	-0.0211	-0.0155
	(-1.615)	(-1.564)	(-0.544)	(-0.430)
immunization rate	-0.673***	-0.499***	-0.656***	-0.496***
	(-4.412)	(-3.611)	(-4.069)	(-3.401)
institution quality	0.0329	0.0396	0.0279	0.0346
	(0.993)	(1.321)	(0.620)	(0.899)
GDP per capita	-0.358***	-0.234***	-0.304***	-0.186**
	(-6.372)	(-4.134)	(-4.182)	(-2.649)
urban population	-0.0946	-0.0950	-0.0381	-0.0409
	(-0.908)	(-0.847)	(-0.374)	(-0.381)
population density	0.0115	0.0340	-0.0300	0.000896
	(0.390)	(1.269)	(-1.215)	(0.0355)
Constant	3.678***	1.682**	1.173	-0.556
	(4.267)	(2.234)	(1.274)	(-0.612)
year dummies	yes	yes	yes	yes
quintile dummies	yes	yes	yes	yes
Observations	300	300	330	330
R-squared	0.87	0.78	0.86	0.79

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

3.5.2 Heterogeneity in the health effect of air pollution (intra country inequalities)

In the previous subsection, we found that pollution is in part responsible to health inequality between countries. This section extends these results and explores whether environmental degradation may contribute to within country income related health inequalities. It presents the results obtained from the estimation of equation (3.4.2) and these results are summarized in Table 3.2. In this table, the coefficients of interest are those of the interaction terms of environmental variables and quintile dummies (λ_i).

These coefficients are higher for poor quintiles as compare to those of richest quintiles. In addition, they are negative and statistically significant for richest quintiles and not significant for poorest quintiles. These results show that, environmental degradation degrades more the health outcomes of poorest quintiles than it worsens those of the richest quintiles. This heterogeneity in the health effect of air pollution increases income related health inequality within country. These results are in conformity with our theoretical hypothesis and arguments. Besides these findings, all the variables already analysed in previous subsection present the correct signs and are statistically significant.

Table 3. 2: Impact of air pollution on health inequalities within countries

	Dependent variables			
	Sulphur dioxide emission (SO2)		Particulate Matter (PM10)	
Independent variables	(1) Child mortality	(2) Inf. mortality	(3) Child mortality	(4) Inf. mortality
air pollution	0.129***	0.116***	0.208**	0.187**
	(3.395)	(3.459)	(2.352)	(2.238)
(air pollution)x(quintile 2)	-0.0321	-0.0209	-0.0176	-0.0181
	(-1.307)	(-0.969)	(-0.412)	(-0.426)
(air pollution)x(quintile 3)	-0.0479**	-0.0592**	-0.0511	-0.0394
	(-2.021)	(-2.268)	(-0.978)	(-0.694)
(air pollution)x(quintile 4)	-0.0549*	-0.0591	-0.0979	-0.0794
	(-1.938)	(-1.567)	(-1.278)	(-0.938)
(air pollution)x(quintile 5)	-0.0823*	-0.0934*	-0.192**	-0.128
	(-1.706)	(-1.680)	(-2.256)	(-1.514)
fertility rate	0.505***	0.323***	0.665***	0.478***
	(4.884)	(3.063)	(5.086)	(3.500)
schooling	-0.0776*	-0.0754*	-0.000414	-0.00177
	(-1.848)	(-1.928)	(-0.00939)	(-0.0425)
immunization rate	-0.658***	-0.481***	-0.680***	-0.512***
	(-4.337)	(-3.571)	(-4.204)	(-3.470)
institution quality	0.0321	0.0387	0.0290	0.0353
	(0.956)	(1.277)	(0.639)	(0.904)
GDP per capita	-0.357***	-0.232***	-0.303***	-0.185**
	(-6.325)	(-4.133)	(-4.095)	(-2.586)
urban population	-0.0978	-0.0988	-0.0313	-0.0361
	(-0.948)	(-0.893)	(-0.302)	(-0.329)
population density	0.00958	0.0316	-0.0264	0.00340
	(0.329)	(1.202)	(-1.028)	(0.129)
Constant	4.242***	2.286***	0.740	-0.874
	(4.871)	(2.900)	(0.737)	(-0.866)
year dummies	yes	yes	yes	yes
quintile dummies	yes	yes	yes	yes
Observations	300	300	330	330
R-squared	0.87	0.78	0.87	0.79

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

3.5.3 Roles of political institutions in the health inequality effect of pollution

We have previously found that pollution is harmful for population health and the poorest income classes are those that suffer more from this effect. This section is devoted to the roles played by political institutions regarding this effect of air pollution on health inequality. It shows the results obtained from the estimation of equation (3.4.3) and the findings are presented in table 3.3.

In this table, we are interested by the coefficients of the interaction terms of environmental variables, institutions and quintile dummies (γ_i). These coefficients are higher for richest quintiles than poorest quintiles. That result demonstrates that good political institutions mitigate more the health effect of air pollution for the poorest quintiles than they do for richest income classes. We can conclude that political institutions contribute to reduce the health inequalities created by environmental degradation by mitigating its impact on the poor.

To test the robustness of our result to the choice of institutional indicator, we replace our institutional variable (military in politics) by successively bureaucracy quality, corruption, law and order, democracy accountability, internal conflict, and freedom status indices. The results obtained are presented in Appendix 3.5 and they remain unchanged, namely, the coefficients of the interaction terms of environmental variables, institutions and quintile dummies (γ_i) are higher for richest income quintiles as compared to poorest ones.

Table 3. 3: Social protection role of political institutions

	Dependent variable			
	Sulphur dioxide emission (SO2)		Particulate Matter (PM10)	
	(1)	(2)	(3)	(4)
Independent variables	Inf. mortality	Child mortality	Inf. mortality	Child mortality
air pollution	0.280***	0.319***	0.0718	0.0963
	(2.996)	(3.086)	(0.467)	(0.558)
(air pollution)x(quintile 2)	0.0102	0.0116	0.129	0.156
	(0.180)	(0.232)	(1.431)	(1.599)
(air pollution)x(quintile 3)	-0.140	-0.109	0.0995	0.125
	(-1.596)	(-1.418)	(1.022)	(1.307)
(air pollution)x(quintile 4)	-0.107	-0.107	0.0323	0.0447
	(-1.157)	(-1.307)	(0.220)	(0.336)
(air pollution)x(quintile 5)	-0.290***	-0.281***	0.0266	-0.00222
	(-3.090)	(-3.085)	(0.229)	(-0.0189)
(institution)x(quintile 2)	-0.117	-0.162	0.241*	0.287**
	(-0.557)	(-0.788)	(1.758)	(2.072)
(institution)x(quintile 3)	0.272	0.201	0.217	0.282*
	(1.009)	(0.829)	(1.386)	(1.903)
(institution)x(quintile 4)	0.146	0.162	0.162	0.222
	(0.542)	(0.645)	(0.621)	(0.939)
(institution)x(quintile 5)	0.664**	0.675**	0.219	0.285
	(2.282)	(2.265)	(0.908)	(1.209)
(institution)x(air pollution)	-0.0388**	-0.0449**	0.0401	0.0396
	(-2.191)	(-2.274)	(0.749)	(0.685)
(institution)x(air pollution)x(quintile 2)	-0.00852	-0.0121	-0.0585**	-0.0688**
	(-0.500)	(-0.719)	(-2.033)	(-2.255)
(institution)x(air pollution)x(quintile 3)	0.0236	0.0179	-0.0568	-0.0710**
	(1.142)	(0.957)	(-1.636)	(-2.120)
(institution)x(air pollution)x(quintile 4)	0.0139	0.0144	-0.0438	-0.0551
	(0.684)	(0.736)	(-0.766)	(-1.047)
(institution)x(air pollution)x(quintile 5)	0.0543**	0.0550**	-0.0581	-0.0730
	(2.532)	(2.478)	(-1.095)	(-1.386)
fertility rate	0.328***	0.504***	0.471***	0.661***
	(3.035)	(4.552)	(3.601)	(5.274)
schooling	-0.107***	-0.107***	-0.0261	-0.0212
	(-2.904)	(-2.804)	(-0.613)	(-0.473)
immunization rate	-0.538***	-0.732***	-0.542***	-0.724***
	(-4.374)	(-4.650)	(-4.176)	(-4.859)
GDP per capita	-0.241***	-0.371***	-0.179**	-0.299***
	(-4.560)	(-6.783)	(-2.411)	(-3.841)
institution quality	-0.434*	-0.518**	-0.110	-0.112
	(-1.945)	(-2.118)	(-0.455)	(-0.441)
urban population	-0.0944	-0.0953	-0.0272	-0.0177
	(-0.939)	(-1.016)	(-0.238)	(-0.157)
population density	0.0551*	0.0358	0.0137	-0.0141
	(1.765)	(1.047)	(0.487)	(-0.500)
Constant	4.588***	6.981***	-0.318	1.295
	(3.175)	(3.996)	(-0.289)	(1.052)
year dummies	yes	yes	yes	yes
quintile dummies	yes	yes	yes	yes
Observations	300	300	330	330
R-squared	0.80	0.88	0.79	0.87

3.6. Robustness checks

In the previous section we showed that air pollution is more disastrous for poor people's health (poor income quintiles health) than that of rich people (rich income quintile), and therefore increases income related health inequality within population. One could argue that these results suffer from at least three drawbacks. First, because environmental variable is not disaggregated by asset quintile, we did not take into account country fixed effects and this could bias our results. The second problem also comes from the structure of our data. In fact, the dependent variables (health variables) are more disaggregated than the variables of interest (environment and institution variables), and that may downward-bias the standard deviations because of Moulton bias (Moulton, 1987 and 1990). Moulton (1990) demonstrated that if the disturbances are correlated within the groupings that are used to merge aggregated with micro data, the standard errors from Ordinary Least Squares (OLS) are seriously biased downward. Third, one could argue that we assessed the effect of environment on health inequality, but we did not use explicitly any health inequality indicator. To solve for this, we replace health indicator by the range, more precisely we use as alternative dependent variable the logarithmic form of the ratio of the first quintile of mortality rates to those of the fifth quintile. This indicator is largely used in the literature to measure health inequality (Wagstaff et al. 1991; Levine et al. 2001). That is, all the variables are expressed in country level.

$$health_{jt} = X'_{jt}\beta + \delta environment_{jt} + \mu_i + \varepsilon_{jt} \qquad (3.6.1)$$

The results obtained from the estimation of this equation with fixed effect are presented in Appendix 3.6. The coefficients of environment indicators are positive and statistically significant showing that air pollution increases mortality gap between rich and poor asset groups in a given country, and this confirms our previous results.

To verify the role played by institutions quality in this effect of pollution on health inequality, we add to the previous equation the interaction term of environment and institutional variables and we obtain the following equation.

$$health_{jt} = X'_{jt}\beta + \delta environment_{jt} + \psi(environment_{jt} * institution) + \varepsilon_{jt} \quad (3.6.2)$$

We also estimate this equation with fixed effect and the results are summarized in Appendix 3.7. The coefficients of environment indicators remain positive and statistically significant, and those of the interaction terms are negative and significant.

These results confirm our previous findings, namely good political institutions contribute to reduce the health inequalities created by environmental degradation. However, as argued by Wagstaff et al. (1991), the range overlooks what is going on in the intermediate groups. The gap between the first and the fifth quintiles might, for example, remain unchanged, but the extent of inequality between the intermediate quintiles might well be diminishing (or increasing). In addition, it does not take into account the sizes of the indicators being compared. This can lead to misleading results when comparisons are performed over time or across countries.

This can be solved by using as health inequality indicator, the concentration index of mortality rates. This indicator is commonly used to represent health inequality, because of its affinity with the Gini coefficient, its visual representation by means of the Concentration Curve and the ease with which it can be decomposed. It can be calculated at individual level as well as socioeconomic group level (income quintile level). It cannot be lower than -1 and higher than 1. A negative (positive) value of the concentration index of mortality rates designates a more concentrated mortality within poor (rich) people. A zero value indicates an equal distribution of mortality according to income quintiles.

As argued by Erreygers (2006), this indicator is far from perfect. The first criticism is from Wagstaff (2005). He argues that if the health variable is binary, the bounds of the Concentration Index depend upon the mean of the health variable. The bounds turn out to be much wider for populations with a low mean than for populations with a high mean. To address this issue, he proposes to divide the health Concentration Index by its upper bound. According to Erreygers (2006) Wagstaff procedure exaggerates the correction it applies to the index and to its bounds, and an alternative solution has been formulated originally by Wagstaff et al. (1991). This indicator called Generalized health Concentration Index is obtained by multiplying the health Concentration Index by the average health level.

We use in this section as alternative health inequality indicator in equations (3.6.1) and (3.6.2) the Generalized Concentration Index of mortality rates. The results obtained with fixed effects estimator are presented in Appendix 3.8 and Appendix 3.9 respectuvely. They remain similar to previous results.

3.7. Concluding remarks

This chapter extends economic literature on the association between environment and health by investigating the responsibility of air pollution in the explanation of health inequalities both between and within developing countries. It examines also the importance of the role played by good political institutions in this effect.

We argue that population belonging to poorest income quintiles are those likely to suffer more from environmental degradation, because they receive the highest exposure, and this exposure then exercises larger effects on their health than it does on the average population. Furthermore, richest communities have more prevention than poorest and have more access to medical care when they are sick from pollution.

In countries with good political institutions, this heterogeneity in the health effect of pollution may be mitigated since these institutions favour universal health policy issues, information and advices about hygiene, and health infrastructures building.

Globally, our econometric results corroborate these theoretical arguments and hypothesis about the positive association between air pollution and income related health inequalities. In addition, our empirical results confirm the significant role played by democratic institutions in protecting poor population from environmental degradation.

These important findings raise some policy implications. First, to be effective, health policies should not be based only on average health of a given population, but also on its distribution. In addition, differential distribution of health effects of pollution should be considered alongside differential distribution of the benefits related to the emission sources. Indeed, those who pollute more in a population, such as car ownership may compensate those who bear the adverse effect by paying a tax. Finally, improving political institutions is not only important for economic growth, but it is also essential for population wellbeing.

This study could be extended in many ways. Firstly, a limit of this work is doubtless the unavailability of environmental data varying across income quintiles. This kind of data takes into account the differential of exposure. Future works on this topic should solve for this and test our hypothesis with more accurate data. Researchers may also use other environmental and health indicators to verify our hypothesis. We focus only on developing countries. It will be interesting to extend our results by testing whether they may be generalized for developed countries or compare them across different geographical regions.

APPENDICES 3

Appendix 3.1: determinants of health inequality in the literature

Human capital in general and particularly population health is one of the most important factors in economic development and a healthy labour force is essential for the development. Average health in a population alone is not sufficient, since it could hide a large disparity in health. Both mean health and health distribution should be considered. In economic literature, many factors have been underlined as health inequality determinants.

- *Income*: The first determinant of inequalities is income and income inequality. In fact, countries with higher income are more able to implement effective health policies and provide high quality health services for all the population everywhere. A simple example is the availability of hospitals and physicians in developed countries. In poor countries, high quality health services are concentrated in big cities and only a small amount of population has access. Income inequality also explains health disparities because individual income level affects health through its effect on consumption behaviour (against malnutrition), drinking water quality (hygiene), clothes, housing, preventive cure and information. Inequality in health, then, increases difference in health outcomes within individuals. Ourti et al (2009) show that, theoretically, both income growth and reductions in social inequalities in health and income can be achieved only under very specific conditions concerning the type of growth and the income responsiveness of health. According to them, income growth and income inequality have a direct and indirect effect on income-related health inequality. The sign of the direct effects can be derived a priori, but not of the indirect effects. The expected

direct effect of proportional income growth depends crucially on the slope of the income elasticity. If this elasticity is rising (decreasing) with income on average, income-related health inequality will increase (decline). With respect to the direct effect of changes in income inequality, they find increasing (decreasing) income-related health inequality in case of on average pro-rich (pro-poor) evolving income inequality in combination with an income elasticity that increases (decreases) with income on average. They investigate empirically whether these conditions were met in Europe in the 1990s using panel data from the European Community Household Panel. Their results show that in most countries, the income elasticity of health was positive and increases with income, and that income growth was not pro-rich in most European countries, resulting in small or negligible reductions in income inequality. The combination of both findings explains the modest increases in income-related health inequality in the majority of countries. Tubeuf (2009) used an innovative methodology to measure income related health inequalities using the concentration index, and investigated the relationships between income, income inequality, various social determinants, and health. His results show an income-related health inequality favouring individuals with a higher income. Moreover, income level, supplementary private health insurance, education level, and social class account for the main contributions to inequality. Therefore reducing income inequality is not sufficient to lower income-related health inequalities and needs to be supplemented with the reduction of the relationship between income and health and the reduction of income inequality over socioeconomic status.

- *Education*: The distribution of education also may provoke health inequality within a population, (Grossman, 1972; Schultz, 2002) since education influences consumption behaviour and improves knowledge about health care (hygiene, contraception). In addition, more educated people know about and ask for beneficial health procedure such as quitting

smoking; getting flu shots; wearing seat belts and driving a car with airbags; eating fruits and vegetables; exercising regularly. Rahkonen et al. (1995) compare whether the relationship between social class and health is similar among young men and women at different age groups in Britain and Finland and examine at what age social class differences in self-reported health and illness among young adults emerge. They found that the best discriminator of differences in ill-health among young adults both in Finland and Britain was education.

- *Distribution of health care services*: The disparities in the availability of health care services across regions of a country induce health inequality since some populations easily have access to health services and other cannot have access to such services when they need them. Using cross-sectional data from 31 provinces, Fang et al (2010) measured the degree of regional health inequality in China and identified its determinants through canonical correlation analysis. They found that there existed distinct regional disparities in health in China, which were mainly reflected in Maternal & Child Health and Infectious Diseases, not in the average life expectancy. They also showed that the regional health inequality was associated with not only the distribution of wealth, but also the distribution of health resources and primary health care services.

- *Place and neighbouring*: Place and neighbouring also may influence health inequality. Using place as a relational space linked to where people live, work and play, Bernard et al. (2007) conceptualise the nature of neighbourhoods as they contribute to the local production of health inequalities in everyday life. They propose that neighbourhoods essentially involve the availability of, and access to, health-relevant resources in a geographically defined area. They argue that such availability and access are regulated according to four different sets of rules: proximity, prices, rights, and informal reciprocity. Their theoretical framework

supposes that these rules give rise to five domains, the physical, economic, institutional, local sociability, and community organisation domains which cut across neighbourhood environments through which residents may acquire resources that shape their lifecourse trajectory in health and social functioning.

- *Health insurance*: Countries with effective health insurance system are more likely to have less disparity in health outcomes. In fact, health insurance increases the access to health care services by reducing the burden of health cost and by improving prevention. In his analysis Tubeuf (2009) shows that health insurance contributes to reduce health inequality.

Appendix 3.2: list of countries in the regression sample

country name	Year		country name	Year
Armenia	2000		Madagascar	1997
Benin	1996, 2001		Mali	1995, 2001
Burkina Faso	1992, 1998, 2003		Mozambique	1997, 2003
Bangladesh	1996, 1999, 2004		Mauritania	2000
Bolivia	1998, 2003		Malawi	1992, 2000
Brazil	1996		Namibia	1992, 2000
Central African Republic	1994		Niger	1998
Côte d'Ivoire	1994		Nigeria	1990, 2003
Cameroon	1991, 1998, 2004		Nicaragua	1997, 2001
Colombia	1995, 2000, 2005		Nepal	1996, 2001
Comoros	1996		Pakistan	1990
Dominican Republic	1996, 2002		Peru	1996, 2000
Egypt	1995, 2000		Philippines	1998, 2003
Eritrea	1995		Paraguay	1990
Ethiopia	2000		Rwanda	2000
Gabon	2000		Senegal	1997
Ghana	1993, 1998, 2003		Chad	1996, 2004
Guinea	1999		Togo	1998
Guatemala	1995, 1998		Turkmenistan	2000
Haiti	1994, 2000		Turkey	1993, 1998
Indonesia	1997, 2002		Tanzania	1996, 1999, 2004
India	1992, 1998		Uganda	1995, 2000
Jordan	1997		Uzbekistan	1996
Kazakhstan	1995, 1999		Vietnam	1997, 2002
Kenya	1993, 1998, 2003		Yemen	1997
Kyrgyzstan	1997		South Africa	1998
Cambodia	2000		Zambia	1996, 2001
Morocco	1992, 2003		Zimbabwe	1994, 1999

Appendix 3.3: Summary Statistics by quintile

Variables	Obs	Mean	Std. Dev.	Min	Max
Full sample					
Infant mortality (a)	380	72.13	33.75	11.90	187.70
Child mortality (b)	380	113.80	67.00	14.20	354.90
Fertility rate (c)	380	4.55	1.80	1.20	8.50
Female educational attainment (d)	380	50.44	31.94	0.50	99.80
Poorest quintile (based on an "asset index")					
Infant mortality (a)	76	86.88	31.32	32.00	187.70
Child mortality (b)	76	140.08	62.82	39.10	297.90
Fertility rate (c)	76	5.92	1.48	2.20	8.50
Female educational attainment (d)	76	29.15	25.98	0.50	98.70
Second quintile					
Infant mortality (a)	76	82.62	32.71	23.80	152.30
Child mortality (b)	76	132.33	69.25	27.30	354.90
Fertility rate (c)	76	5.14	1.55	1.80	8.20
Female educational attainment (d)	76	39.24	29.75	1.00	99.50
Third quintile					
Infant mortality (a)	76	75.91	34.14	19.70	157.20
Child mortality (b)	76	120.08	69.44	23.50	348.30
Fertility rate (c)	76	4.68	1.65	1.40	7.80
Female educational attainment (d)	76	48.38	30.98	1.50	99.80
Fourth quintile					
Infant mortality (a)	76	65.64	32.17	11.90	142.00
Child mortality (b)	76	102.63	64.63	14.20	314.90
Fertility rate (c)	76	4.02	1.61	1.50	7.20
Female educational attainment (d)	76	59.09	29.71	4.80	99.60
Richest quintile					
Infant mortality (a)	76	49.58	24.51	13.80	97.20
Child mortality (b)	76	73.88	45.93	15.80	183.70
Fertility rate (c)	76	2.96	1.15	1.20	6.20
Female educational attainment (d)	76	76.34	20.13	27.00	99.80

Notes :

(a) Infant mortality: number of deaths to children under twelve months of age per 1,000 live births, based on experience during the ten years before the survey.

(b) Child mortality: number of deaths to children under five years of age per 1,000 live births, based on experience during the ten years before the survey.

(d) Fertility rate: average number of births a woman could expect to have during her lifetime if she followed the levels of fertility currently observed at every age. The TFR is calculated as the sum of average annual age specific fertility rates for all reproductive age groups (usually 15-49 years) in the three years before the survey.

(c) Female educational attainment: percent of women aged 15-49 years who had completed the fifth grade

Appendix 3.4. Data characteristics and sources

Variables	mean	min	max	Coef. var.	Obs.	characteristics	Sources
Infant mortality rate	74,65	22,1	147,4	0,39	95	Number of deaths to children under twelve months of age per 1,000 live births, based on experience during the ten years before the survey.	Gwatkin et al. (2007)
Under five mortality rate	118,6	25,7	302,6	0,51	95	Number of deaths to children under five years of age per 1,000 live births, based on experience during the ten years before the survey.	Gwatkin et al. (2007)
Sulphur dioxide emission (SO2)	239,86	0,53	2926,53	2,18	73	sulphur dioxide emission	Stern (2005)
Particulate Matter (PM10)	77,99	7,3	225,86	0,62	82	particulate matter less than 10 µm aerodynamic diameter	WDI 2007
urban population percentage	38,43	11,4	80,1	0,47	95	Proportion of urban population	WDI 2007

Variables	mean	min	max	Coef. var.	Obs.	characteristics	Sources
population density	110,19	1,85	1156,4	1,75	95	Population density	WDI 2007
fertility rate	5,88	2,2	8,5	0,26	95	Average number of births a woman could expect to have during her lifetime if she followed the levels of fertility currently observed at	Gwatkin et al. (2007)
schooling	30,25	0	99,1	0,92	94	Percent of women aged 15-49 years who had completed the fifth grade.	Gwatkin et al. (2007)
GDP per capita	815,38	120,11	4286,5	1,03	95	Gross Domestic Product per capita	WDI 2007

Appendix 3.5: Robustness checks : Social protection role of political institutions.

	Dependent variables											
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Independent variables	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.
air pollution	0.153**	0.153***	0.184**	0.196**	0.0339	0.0110	0.120*	0.114	0.0509	0.104	0.223**	0.259***
	(2.683)	(2.862)	(2.225)	(2.654)	(0.447)	(0.147)	(1.953)	(1.632)	(0.498)	(0.829)	(2.665)	(2.936)
(air pollution)x(quintile 2)	0.0341	0.0290	0.0596	0.0627*	0.0984***	0.0846**	0.0760**	0.0607*	0.119*	0.107	0.0602	0.00915
	(0.758)	(0.963)	(1.351)	(1.712)	(3.565)	(2.437)	(2.048)	(1.886)	(1.860)	(1.489)	(0.836)	(0.149)
(air pollution)x(quintile 3)	-0.0580	-0.0320	-0.0224	0.00145	0.0447	0.0865*	-0.0640	-0.0332	-0.0908	-0.0568	-0.128*	-0.102*
	(-1.049)	(-0.621)	(-0.306)	(0.0244)	(0.801)	(1.877)	(-0.807)	(-0.500)	(-1.110)	(-0.798)	(-2.004)	(-1.772)
(air pollution)x(quintile 4)	-0.137*	-0.0948	-0.116	-0.0940	-0.0262	0.0497	-0.150**	-0.106*	-0.119	-0.119	-0.163*	-0.151*
	(-1.842)	(-1.293)	(-0.772)	(-0.795)	(-0.420)	(0.943)	(-2.152)	(-1.964)	(-1.146)	(-1.300)	(-1.905)	(-1.859)
(air pollution)x(quintile 5)	-0.245***	-0.181**	-0.234	-0.177	0.00118	0.0739	-0.116	-0.0857	-0.379***	-0.317***	-0.457***	-0.417***
	(-3.602)	(-2.444)	(-1.571)	(-1.365)	(0.00734)	(0.508)	(-0.759)	(-0.614)	(-3.361)	(-3.024)	(-4.204)	(-4.459)
(institution)x(quintile 2)	-0.355	-0.387***	-0.364**	-0.430***	-0.725***	-0.694***	-0.400***	-0.385**	-0.518**	-0.520*	-0.117	-0.0543
	(-1.542)	(-2.958)	(-2.369)	(-2.777)	(-4.960)	(-2.950)	(-2.907)	(-2.406)	(-2.121)	(-1.769)	(-1.013)	(-0.489)
(institution)x(quintile 3)	0.0590	-0.0332	-0.164	-0.221	-0.605*	-0.759***	0.00889	-0.0641	0.126	0.0423	0.115	0.0936
	(0.228)	(-0.141)	(-0.585)	(-0.975)	(-1.940)	(-3.107)	(0.0352)	(-0.302)	(0.438)	(0.163)	(1.250)	(1.074)
(institution)x(quintile 4)	0.623	0.370	0.264	0.182	-0.0138	-0.467	0.363	0.199	0.238	0.273	0.170	0.162
	(1.456)	(0.954)	(0.404)	(0.368)	(-0.0343)	(-1.523)	(1.403)	(0.972)	(0.627)	(0.866)	(1.328)	(1.350)
(institution)x(quintile 5)	1.124**	0.764*	0.644	0.434	-0.445	-0.813	0.0739	0.00179	1.080**	0.900**	0.559***	0.522***
	(2.631)	(1.832)	(0.917)	(0.725)	(-0.430)	(-0.882)	(0.126)	(0.00334)	(2.608)	(2.278)	(3.398)	(3.666)
(institution)x(air pollution)x(quintile 2)	-0.0252	-0.0265***	-0.024**	-0.0296**	-0.060***	-0.0584***	-0.0312***	-0.0298**	-0.0411**	-0.0406*	-0.0103	-0.00561
	(-1.531)	(-2.956)	(-2.077)	(-2.359)	(-4.785)	(-2.907)	(-2.805)	(-2.274)	(-2.186)	(-1.782)	(-1.142)	(-0.633)
(institution)x(air pollution)x(quintile 3)	0.0110	0.00482	-0.00615	-0.0102	-0.0507**	-0.0633***	0.00237	-0.00449	0.00855	0.00225	0.00746	0.00583
	(0.562)	(0.279)	(-0.296)	(-0.584)	(-2.057)	(-3.212)	(0.124)	(-0.277)	(0.413)	(0.120)	(1.077)	(0.850)
(institution)x(air pollution)x(quintile 4)	0.0563*	0.0380	0.0268	0.0203	-0.00539	-0.0410*	0.0300	0.0171	0.0161	0.0170	0.0115	0.0105
	(1.765)	(1.328)	(0.553)	(0.558)	(-0.180)	(-1.814)	(1.544)	(1.114)	(0.597)	(0.740)	(1.175)	(1.138)
(institution)x(air pollution)x(quintile 5)	0.0956***	0.0678**	0.0541	0.0377	-0.0326	-0.0649	0.00793	0.000878	0.0813***	0.0666**	0.0430***	0.0392***
	(3.081)	(2.261)	(1.006)	(0.827)	(-0.417)	(-0.927)	(0.177)	(0.0217)	(2.754)	(2.372)	(3.386)	(3.558)
(institution)x(air pollution)	-0.0188	-0.0147	-0.0279	-0.0274	0.0344	0.0507	-0.000535	0.00673	0.0192	0.00839	-0.0109	-0.0134
	(-0.644)	(-0.538)	(-1.044)	(-1.142)	(1.032)	(1.488)	(-0.0283)	(0.326)	(0.660)	(0.248)	(-1.373)	(-1.606)
fertility rate	0.353***	0.528***	0.336***	0.515***	0.419***	0.694***	0.338***	0.520***	0.342***	0.524***	0.360***	0.546***
	(3.085)	(4.843)	(3.108)	(4.938)	(3.540)	(5.686)	(2.937)	(4.565)	(2.977)	(4.807)	(3.542)	(5.887)

	Dependent variables											
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Independent variables	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.	inf. mort.	child mort.
schooling	-0.0862**	-0.0859*	-0.0744*	-0.0765*	-0.0601*	-0.0747*	-0.0833**	-0.0863*	-0.0904**	-0.0864*	-0.0870**	-0.0853*
	(-2.119)	(-1.943)	(-1.891)	(-1.818)	(-1.680)	(-1.985)	(-2.035)	(-1.948)	(-2.092)	(-1.860)	(-2.114)	(-2.009)
immunization rate	-0.380***	-0.583***	-0.47***	-0.650***	-0.228*	-0.339**	-0.428***	-0.601***	-0.402***	-0.603***	-0.343**	-0.524***
	(-2.864)	(-3.780)	(-3.513)	(-4.293)	(-1.831)	(-2.243)	(-3.099)	(-3.808)	(-2.790)	(-3.541)	(-2.398)	(-3.173)
GDP per capita	-0.219***	-0.342***	-0.23***	-0.353***	-0.235***	-0.328***	-0.237***	-0.357***	-0.220***	-0.350***	-0.242***	-0.370***
	(-3.303)	(-4.851)	(-4.014)	(-6.142)	(-4.226)	(-5.305)	(-4.231)	(-6.093)	(-3.865)	(-5.953)	(-4.328)	(-6.719)
institution quality	-0.225	-0.154	-0.268	-0.266	0.395	0.599	0.0280	0.0887	0.249	0.0986	-0.180	-0.220*
	(-0.575)	(-0.420)	(-0.772)	(-0.882)	(0.886)	(1.340)	(0.113)	(0.329)	(0.581)	(0.198)	(-1.668)	(-1.988)
urban population	-0.104	-0.106	-0.102	-0.104	-0.0642	-0.0570	-0.0913	-0.0906	-0.101	-0.0939	-0.0918	-0.0887
	(-0.843)	(-0.896)	(-0.879)	(-0.952)	(-0.649)	(-0.574)	(-0.832)	(-0.842)	(-0.953)	(-0.942)	(-0.813)	(-0.825)
population density	0.0377	0.0153	0.0349	0.0132	0.0393	0.0377	0.0278	0.00991	0.0213	0.00474	0.0203	-0.00226
	(1.390)	(0.497)	(1.308)	(0.443)	(1.312)	(1.134)	(1.059)	(0.317)	(0.680)	(0.140)	(0.678)	(-0.0690)
Constant	2.240*	4.105***	2.953**	4.893***	-0.0392	0.726	2.121*	3.812***	1.201	3.752*	3.484**	5.803***
	(1.916)	(3.652)	(2.319)	(3.903)	(-0.0349)	(0.609)	(1.878)	(2.964)	(0.779)	(1.987)	(2.632)	(4.056)
Institution indicators	Bureaucracy quality		corruption index		freedom status		democracy accountability		law and order		internal conflict	
year dummies	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
quintile dummies	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
Observations	300	300	300	300	360	360	300	300	300	300	300	300
R-squared	0.80	0.88	0.79	0.87	0.74	0.84	0.79	0.87	0.79	0.87	0.79	0.87

***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

Appendix 3.6: Effect of air pollution on health inequality

	Dependent variable: log of the Ratio of poorest quintile to richest quintile of infant mortality rate (Q1/Q5)	
VARIABLES	(1)	(2)
Sulphur dioxide emission (SO2)	0.0541***	
	(4.045)	
Particulate Matter (PM10)		0.968***
		(5.412)
fertility rate ratio (Q1/Q5)	0.838***	0.760***
	(8.662)	(3.411)
Schooling ratio (Q1/Q5)	1.086	3.883**
	(1.330)	(2.493)
Schooling	68.78***	-29.04
	(4.057)	(-1.550)
Institution quality	-0.159***	0.0720
	(-3.688)	(1.301)
GDP per capita	-4.016***	0.668
	(-8.938)	(1.597)
Immunization ratio (Q1/Q5)	3.294***	1.688*
	(11.45)	(1.690)
Constant	18.70***	-9.814***
	(7.569)	(-2.880)
Fixed effects	yes	yes
Quintiles dummy	yes	yes
Observations	60	66
R-squared	0.94	0.84

Note: ***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

Appendix 3.7: Role of political institutions in the effect of air pollution on health inequality

	Dependent variable: log of the Ratio of poorest quintile to richest quintile of infant mortality rate (Q1/Q5)			
VARIABLES	(1)	(2)	(3)	(4)
Sulphur dioxide emission (SO2)	0.371*	0.967***	1.100***	0.305***
	(1.991)	(22.92)	(5.070)	(5.538)
(Sulphur dioxide emission)x(institution)	-0.0783*	-0.286***	-0.265***	-0.0297***
	(-1.707)	(-21.10)	(-5.145)	(-5.442)
Institution quality	-1.193*	-4.066***	-4.054***	-0.422***
	(-1.965)	(-22.08)	(-5.565)	(-6.242)
fertility rate ratio (Q1/Q5)	1.188***	1.737***	0.820***	1.089***
	(4.454)	(39.10)	(11.23)	(10.87)
Schooling ratio (Q1/Q5)	1.601*	4.732***	0.612	1.809***
	(1.914)	(22.33)	(1.188)	(3.694)
Schooling	78.34***	39.00***	15.19	34.35***
	(5.885)	(13.63)	(1.504)	(4.555)
GDP per capita	-5.412***	-4.361***	-0.0215	-3.888***
	(-5.172)	(-40.11)	(-0.0559)	(-8.264)
Immunization ratio	4.205***	6.444***	2.572***	3.828***
	(6.187)	(38.85)	(17.13)	(13.25)
Constant	29.58***	28.92***	12.48***	21.92***
	(3.847)	(29.48)	(5.295)	(6.692)
Institution quality indicator	corruption index	Bureaucracy quality	law and order	internal conflict
Fixed effects	yes	yes	yes	yes
year dummies	yes	yes	yes	yes
Observations	60	60	60	60
R-squared	0.95	0.99	0.97	0.96

Note: ***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

Appendix 3.8: Effect of air pollution on health inequality

	Dependent variable: Generalized Concentration index of infant mortality rate	
VARIABLES	(1)	(2)
Sulphur dioxide emission (SO2)	-0.00121***	
	(-5.911)	
Particulate Matter (PM10)		-0.00883***
		(-4.525)
fertility rate ratio (Q1/Q5)	-0.00479***	-0.00815***
	(-2.983)	(-3.935)
schooling concentration index	-1.122	2.619***
	(-1.565)	(4.966)
schooling	-1.396***	0.0986
	(-7.114)	(0.523)
Institution quality	0.00446***	-0.00225***
	(4.148)	(-3.166)
GDP per capita	0.0732***	-0.00914
	(8.958)	(-1.622)
Immunization ratio (Q1/Q5)	-0.0122***	0.0135
	(-3.754)	(1.477)
Constant	-0.390***	0.0650
	(-9.511)	(1.596)
Fixed effects	yes	yes
year dummies	yes	yes
Observations	60	66
R-squared	0.97	0.77

Note: ***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

Appendix 3.9: Role of political institutions in effect of air pollution on health inequality

	Dependent variable: Generalized Concentration index of infant mortality rate			
VARIABLES	(1)	(2)	(3)	(4)
Sulphur dioxide emission (SO2)	-0.00671***	-0.00798***	-0.0285***	-0.00414***
	(-37.62)	(-70.49)	(-14.90)	(-36.68)
(Sulphur dioxide emission)x(institution)	0.00140***	0.00242***	0.00695***	0.000398***
	(29.64)	(65.79)	(14.66)	(38.15)
Institution quality	0.0220***	0.0331***	0.107***	0.00641***
	(39.67)	(66.75)	(14.36)	(39.77)
fertility rate ratio (Q1/Q5)	-0.0115***	-0.0133***	-0.0036***	-0.0105***
	(-32.50)	(-180.1)	(-4.670)	(-64.31)
schooling concentration index	-0.416***	0.344***	-0.444***	-0.0460***
	(-4.366)	(80.10)	(-6.109)	(-3.082)
schooling	-1.455***	-0.626***	0.209***	-0.413***
	(-46.64)	(-91.34)	(4.103)	(-21.31)
GDP per capita	0.0912***	0.0539***	-0.0372***	0.0621***
	(78.93)	(176.8)	(-5.696)	(41.23)
Immunization ratio (Q1/Q5)	-0.0237***	-0.0230***	0.00338**	-0.0191***
	(-42.15)	(-51.19)	(2.444)	(-26.11)
Constant	-0.555***	-0.389***	-0.171***	-0.406***
	(-77.07)	(-146.1)	(-8.083)	(-38.31)
Institution quality indicator	corruption index	Bureaucracy quality	law and order	internal conflict
Fixed effects	yes	yes	yes	yes
year dummies	yes	yes	yes	yes
Observations	60	60	60	60
R-squared	0.98	0.98	0.99	0.99

Note: ***significant at 1%, **significant at 5%, *significant at 10%. t-statistics enter parenthesis.

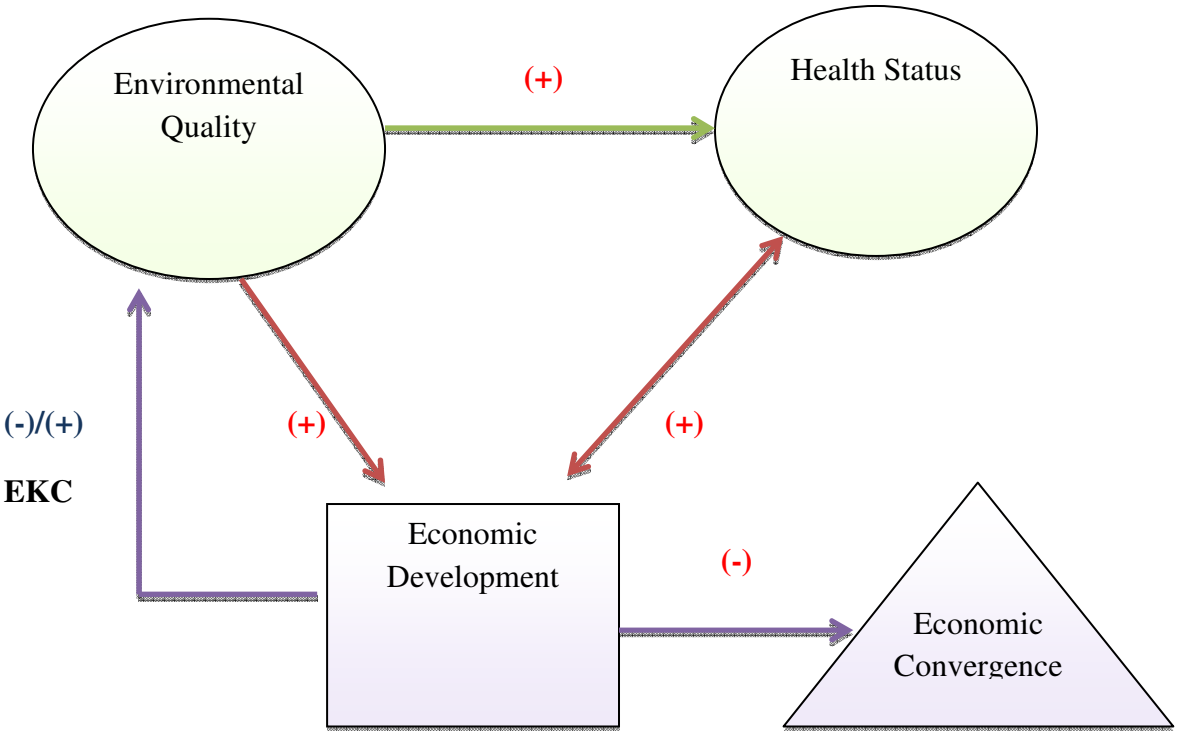
Part II: health, environment and economic growth

Introduction

Economic development or prosperity is an important element of welfare that represents the main objective of all country. It is most widely measured by the Gross Domestic Product (GDP) or Gross National Product (GNP). Policy makers and international development institutions generally build their decisions on the short and long terms improvement of this indicator.

The theoretical and empirical literature on the determinants of economic growth is immense, and a large number of factors have been suggested as fundamental growth determinants. The role of human capital in general and population health in particular as one of these factors, has been investigated. It is generally recognized that good health contributed to economic growth even though some authors rejected this hypothesis (acemoglu, 2007). An important problem about the empirical assessment of the macroecoeconomic health effect is the indicators used in the literature (infant, child, and adult mortality rates, life expectancy, and morbidity). They generally measure only a given aspect of health rather than the overall health outcome in the population. Chapter 4 entitled “Global burden of disease and economic growth” explores this issue, and reassesses the effect of health, measured as global burden of disease, communicable disease, and malaria on economic growth. Figure II.1 presents the link between health and economic development.

Figure II. 1: Interrelationships between environment, health, and economic development



Source : Author

Despite the large literature on the empirical studies on growth determinants, some important potential omitted determinants remain less investigated, such as environmental quality. In fact as shown in Figure II, environmental degradation may affect economic activity directely, or indirectely through the reduction of the level and the productivity of human capital. Air pollutions measured by CO2, SO2, NOx, CO, traffic noise, affect health and leave people unable to work over short or long periods and reduce the productivity of those who work. The assessment of this impact is crucial since it gives additional support on the importance of environmental quality to policy makers.

By illustrating the link between population health and economic growth on one the hand, and the relationship linking economic development to environmental quality on the other hand, Figure II.1 highlights the simultaneous association between environment, health, and

economic development. These interrelationships between the three pillars of sustainable development are less approached in the literature from our knowledge despite the importance of the implications in terms of policy recommendation. Indeed, these interrelationships penalizes the economic progress in developing countries unlike in developed ones, and reduce the ability of poor countries to catch up rich ones. This reduction of the speed of economic convergence is shown in Figure II.1.

Chapter 5, entitled "Interrelationships among health, environment quality and economic activity: What consequences for economic convergence?" deals with these issues by (i) analyzing environmental variables as additional determinant of economic growth, (ii) investigating the interrelationships between environment, health, and economic development, and (iii) assessing the effect of environmental degradation on economic convergence.

On the whole, it is shown that water and air pollutions are important determinants of economic growth, and reduce the ability of poor countries to decrease the gap with developed economies. In addition, the detrimental effect of poor health on economic growth is not rejected.

Chapter 4: Global burden of disease and economic growth[12]

[12] This chapter was written in collaboration with Dr. Martine Audibert and Prof. Pascale Combes Motel. A shorter version was submitted to Journal of Health Economics.

4.1. Introduction

Human capital investments are known for a long time as basic candidates explaining growth performances (e. g. Schultz, 1961). Sen's works on human capabilities and the emergence of AIDS have renewed the interest given to the link between health, welfare, and prosperity. At a microeconomic level, several studies found that poor health have negative effects on economic prosperity and living conditions.[13] At a macroeconomic level, the Commission on Macroeconomics and Health (2001) concluded that diseases raise barriers to economic growth and that countries have to invest in health. Several authors have considered that communicable diseases, among others, had contributed to slow down economic development of low income countries. The latter proposition is still hotly debated as some methodological issues are not satisfactorily addressed (see the comprehensive and critical review of Packard, 2009). For instance, Acemoglu and Johnson (2006), using international data from the epidemiological transition period, find that an increase in life expectancy generated by a decrease in mortality rates had a small positive effect on growth which grows over the post epidemiological transition. The latter was not enough important to compensate for increases in population. This study makes reminiscent previous results of Barlow (1968) with regard to malaria eradication and of Over (1992) with regard to economic effects of AIDS as well. In the same vein, Bell, Bruhns and Gersbach (2006), using an overlapping generations model simulate relaxed effects of AIDS on economic growth in Kenya by 2050.

There are at least three reasons that could explain difficulties to assess health impacts at the macroeconomic level and therefore fuel the debate. First, links between health and development or growth are complex and health effect could, as we saw in Chapter 4, also be

[13] The literature on links between health and economic well-being or prosperity at microeconomic level is abundant. See e.g. Strauss and Thomas (2007) for literature review.

channelled by education levels, the environment, and cultural behaviours as well. When, due to missing adequate indicators, these behaviours are not dealt for in the model, the estimated health effect will be biased or hidden by unobserved heterogeneity (Thomas, 2009; Strauss and Thomas, 2007; Hurd and Kapteyn, 2003). Second, health is subject to measurement errors due to poor measurement facilities such as lack of good equipment and materials for setting appropriate diagnosis, as well as low human resource training, deficient registration, measurement variability over the day (e.g. blood pressure) or the year (e. g. malaria indicators). Third, health status is a rather complex notion that includes several dimensions. Researchers face a wide array of health indicators addressing one specific dimension of health. Consequently, using one or the other is not equivalent.

Partly due to these difficulties of measuring multiple dimensions of health and therefore global health status, macroeconomic effects of health have been more still studied using health indicators such as life expectancy at birth, infant mortality rates, or nutritional status measures. Existing results can be questioned by addressing specifically the choice of health status indicators, which is the subject of this Chapter.

The rest of the chapter is organized as follows. Section 2 is devoted to health measurement issue. Different measures of health indicators used in the growth literature are discussed before exploring the more global one on which the analysis is focused. Section 3 reviews the theoretical and empirical literature on the link between health outcomes and economic performances. The empirical setting and the results are presented in section 4 and 5. Section 6 concludes.

4.2. Looking for a global health indicator

Health measurement is a hard task since, contrary to economic indicators, health is multi-dimensional,[14] and measured with errors. Moreover, researchers, either in a perspective of public health initiatives, health research, or economic health research, have been developing a wide array of health indicators, among which few however are satisfactorily measured (Murray and Frenk, 2008; Murray, 2007).[15] If it is crucial to understand what each indicator measures (Strauss and Thomas, 2008), it is also important to insure that health indicators fit the purposes of studies.

The most commonly used indicators of health conditions at the macroeconomic level are life expectancy at birth and infant mortality rates (Strauss and Thomas, 2008). Those indicators are considered reflecting the general health conditions and supposed to be positively associated with economic growth. It is true that life expectancy at birth is higher and infant mortality lower in richer countries than in poorer countries. Indeed, the correlation between life expectancy at birth and GDP per capita is not systematic as life expectancy is lower (or higher) than expected given GDP per capita in countries like Southern Africa, Gabon or Indonesia (for examples, see Strauss and Thomas, 2008). Per capita incomes have diverged over time while life expectancy and infant mortality have converged (Deaton, 2006; Jack and Lewis, 2009). Life expectancy and infant mortality are inadequate indicators of the population's health in high income countries and for several upper middle income countries where life expectancy at birth is high and infant mortality very low or low. For low and lower middle income countries, those indicators are more adequate due to their poor levels. For that

[14] Whatever the approach chosen (medical, self-assessment or functional) for measuring health, poor health is considering as a deviation between the observed health and a norm. This deviation may occur into either, physical, mental, or social well-being dimension.

[15] For a discussion on the issue and challenge of health measurement, see Mwabu, 2007; Strauss and Thomas, 2008; Murray and Frenk, 2008; Audibert, 2009).

reason, studying the relationships between health and economic development or growth in cross-country studies using infant mortality or life expectancy at birth is not really appropriate, mainly due to the fact that it does not exhibit enough variability in upper middle and high income countries.

As underlined by Jack and Lewis (2009), the effect of a population's health status on national income varies accordingly with the health indicator used. Most health indicators used in the literature capture one dimension of the population health. They either relate to fatal (life expectancy,[16] mortality indicators) or to non-fatal (morbidity indicators) issue of illness (Audibert, 2009). For example, the emergence of HIV/AIDS and its high prevalence (more than 15%) in some southern African countries (Botswana, Lesotho, Namibia, South Africa, Swaziland, Zambia, and Zimbabwe, UNAIDS[17]), have both motivated several studies focusing on their economic effects. But, little evidence of a correlation between HIV/AIDS and GDP per capita was found (Strauss and Thomas, 2008). With the renewed interest for malaria, some authors (Sachs and Malaney, 2002; McCarthy, Wolf and Wu, 2000) have investigated its effect on African countries growth. But, those indicators neither take into account other dimensions of health, such as invalidity, handicap or social consequences, nor multidimensional characteristics of health.

The main thesis of this chapter is that macroeconomic effects of the global health status are accurately caught by the Disability-Adjusted Life Year (DALY) per capita calculated by the World Health Organization (WHO). This indicator was put forward by the World Bank and the World Health Organisation in 1993 (the World Bank, 1993). It represents "a one lost year of healthy life and extends the concept of potential years of life lost due to premature death to

[16] In low income countries, life expectancy is mainly determined by infant mortality, and also in countries where AIDS prevalence is high, by AIDS mortality.

[17] http://www.unaids.org/en/CountryResponses/Regions/SubSaharanAfrica.asp.

include equivalent years of healthy life lost by virtue of being in states of poor health or disability" (WHO, 2008).[18] "The sum of the DALYs across the population represents the burden of disease and can be thought of as a measurement of the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability" (WHO, 2008). DALYs were calculated initially for about one hundred causes and diseases and all over the world and were not updated since 2000. From 2000 to 2004 however, DALYs are also available on a regional basis. DALYs are commonly used in cost-effectiveness analyses but, to the best of our knowledge, have never been used in macroeconomic analyses since DALYs at the country level are only available for 2002 and 2004.

Any indicator, including DALYs, is amenable to criticism with a particular emphasis on weighting (namely age and disease severity) and discounting (e.g. Anand and Hanson, 1998). A large revision has been however implemented, mainly by the Institute of Health Metrics, which is in charge of DALYs updating (Lopez *et al*, 2006). This does not prevent however this indicator from being a serious candidate for representing population global health status, deriving from illness consequences which are taken into consideration in a single indicator.

Appendix 4.1, Appendix 4.2, Appendix 4.3 and Appendix 4.4 present the relationships between different DALY indicators and traditional health measures (Life Expectancy at birth, Infant Mortality Rate and Child Mortality Rate) as well as GDP per capita. It appears clearly that even though there is a positive correlation between DALYs and traditional health indicators, the correlation between them is far from perfect.

[18] The DALYs for each health condition are the sum of the years of life lost (YLL) due to premature mortality and the years lost due to disability (YLD) for incident cases of the health condition. YLL are calculated from the number of deaths at each age multiplied by a global standard life expectancy for each age. YLD is the number of incident cases in a particular period × average duration of the disease × weight factor. The weight factor reflects the severity of the disease on a scale from 0 (perfect health) to 1 (death). For additional information, see WHO, http://www.who.int/healthinfo/global_burden disease/metrics_daly/en/.

4.3. Relationship between health and growth

This chapter lies on the idea of health being a capital: people are endowed an initial stock which can depreciate through time with age but which is the subject of investments (Grossman, 1972; see Mwabu, 2007 for a literature review on the concept of health capital). From such a perspective, Van Zon and Muysken (2005) mention two positive effects of health on economic growth. First, a better health status of population increases labour efficiency; second, human capital accumulation requires "health hours". These effects add to those of Bloom and Canning (2000) who argue that improvements in longevity increase savings and in turn investments; moreover there exists a demographic dividend generated by a decline in child mortality. The effect of health on economic growth has also been the subject of theoretical investigations. One may refer to the augmented Solow model developed and tested by Mankiw et al. (1992). Other authors have included health in optimal Cass-Koopmans like growth models and thereby justified its inclusion in conditional convergence analyses as well: the productivity in the health sector has a positive impact on all steady state variables (Muysken et al. 2003). At last, health investments are taken into account in endogenous growth models *à la Lucas* (1988) with two characteristics: health is produced with decreasing returns whereas human capital is built with increasing returns. Health can either be a complement or a substitute to growth when the effect of health on longevity is internalised (van Zon & Muysken, 2001). Neo-schumpeterian growth models also allow identifying several channels through which population health impacts their long run growth performance. One of these channels puts forward the ability of health improvements to stabilise the gap in living standards relatively to technology leaders (Howitt, 2005).

If at a micro-level, empirical studies found that poor health has an economic effect through several channels (e.g. Audibert 2010), this effect is less evident at a macro-level. The Preston's curve (1975) establishes an upward shifting relationship between life expectancy and national income per capita between 1900 and 1960. The Preston curve does not have sound theoretic foundations. It is at best a correlation which neither gives pieces information on the sense of the causality nor on the different channels through which health may impact economic growth.

These channels may be identified. The first is that healthier people are more productive and supply labour more efficiently. Indeed, they can work harder and longer, and think more clearly. Health status may also improve economic outcomes through its effect on education. Improvements in health raise the motivation to attend high level schooling, since the returns to investments in schooling are valuable over a longer working life. Healthier children and students also have more attendance and higher cognitive functioning, and thus receive a better education for a given level of schooling (see for example, Thuilliez, 2009). Furthermore lower mortality rates and higher life expectancies encourage savings for retirement, and thus raise investment levels and capital per worker. Appendix 4.5 gives a synthesis of some of the main studies that explored the connection between health and economic prosperity. We discuss here some major results.

Some scholars assess empirically how health indicators may influence economic returns in a specific region using individual or household data while others measure the same effect at more aggregated level, between countries or regions. All these studies could be divided according to the health indicators considered. Indeed, a number of studies rely on health inputs whereas others use health outcomes. Health inputs are the physical factors that influence an individual's health and encompass nutrition variables, exposure to pathogens, and availability of medical care (Weil, 2007). Health outcomes are related to the health status

of an individual or a given population. These include health indicators broadly considered such as life expectancy, mortality indicators, the ability to work hard, and cognitive functioning as well as specific illness such as malaria, AIDS/HIV, Guinea worm, cancer, prevalence or incidence, etc.

Researchers generally conclude that population health remains an important predictor of economic outcomes. Life expectancy at birth positively impact economic performances (Barro & Lee, 1994; Cuddington & Hancock, 1994; Barro & Sala-I-Martin, 1995, 2004; Barro, 1996; Sach & Warner, 1997; Bloom & Malaney, 1998; Bloom et al., 2000, 2005, 2009; Arora, 2001; Acemoglu & Johnson, 2007, 2009). Bloom et al. (2004) show that life expectancy has a positive, sizable, and statistically significant effect on aggregate output even when experience of the workforce is controlled for. Sala-i-Martin et al. 2004 departing from the numerous potential explanatory variables in cross-country growth regressions, implement a model selection criterion. The set of explanatory variables which emerges from the analysis includes human capital variables and more especially life expectancy at birth. Acemoglu and Johnson's results (2007) are less conclusive with results indicating that increases in life expectancy have no significant effect on output per capita.[19]

Mortality or survival variables are also used in the literature as overall health outcome indicators that impact economic growth (Hamoudi & Sachs, 1999; Bhargava et al. 2001; Weil, 2007; Lorentzen et al. 2005). Using cross-national and sub-national data, Lorentzen et al. (2005) argue that high adult mortality rates reduce economic growth by shortening time horizons since they favour riskier behaviours, higher fertility rates, and lower investments in physical capital. Other authors are interested in the impact of specific diseases on economic returns. In fact, many diseases like HIV/AIDS and malaria are found to have a negative effect

[19] Even though, Bloom, Canning & Fink (2009) disagree with their results, Acemoglu and Johnson still maintained their position in their 2009 paper.

on the economy (Cuddington & Hancock, 1994; Gallup & al, 1999; Bonnel, 2000; Gallup & Sachs, 2001; Sachs, 2003; Bell, Devarajan and Gerbasch 2003; McDonald & Roberts, 2006; Audibert *et al*., 1998, 1999, 2003, 2006, 2009). McDonald & Roberts (2006) have calculated that the elasticity of economic growth to HIV/AIDS prevalence in Africa is -0.59. Carstensen & Gundlach (2006) found that malaria prevalence causes quantitatively important negative effects on income even after controlling for institutional quality. And, Gallup and Sachs (2001) argued that wiping out malaria from sub-Saharan Africa could increase that continent's per capita growth rate to at least 2.6% a year.

Empirical studies thus do not deliver clear cut effects of health on economic growth: several authors find a negative and significant effect, while others do not. The fact that usual health measures (prevalence, incidence, mortality rate, life expectancy at birth) do not give an accurate measure of the disease burden, may explain that. By including diseases that cause early death but little disability such as diseases that do not cause death but do cause disability, the DALY potentially gives a good indication of the disease burden (WHO, 2008) whatever the main causes of this burden.[20]

4.4. Empirical framework

The analysis of the effect of health on economic growth is based on the augmented neoclassical growth equation, which includes the global health status variable as a regressor combined with initial GDP per capita as catch up variable and other exogenous variables controlling for steady states.

$$y_i = \alpha + \beta Health_i + \mathbf{X'}_i\delta + \epsilon_i$$

[20] 70% of the disease burden is from communicable diseases in Africa, 70% is from non-communicable diseases in high income countries while the part of communicable and non-communicable diseases is equal in middle-income countries (*WHO*, 2008).

Where y_i is the annual growth rate of GDP per capita with subscript i designating the country; $Health_i$ is the global health indicator, DALY; $\mathbf{X}_i$ is the matrix of the k control variables and ε_i is the independently and identically distributed error terms; α, β and δ are parameters to be estimated. Regional dummy variables are included to control for regional specific effects.

4.4.1 Data and variables

The data used in this Chapter cover the period 2000 to 2004. y_i is thus the annual average growth rate on the 2000-2004 period; control variables are average values over the same period. DALYS per capita were calculated for 153 WHO member states (see countries in Appendix 4.11). However, on the studied period, DALYs are available at the country level (*country DALY*) in 2002 and 2004 only. From 2000 to 2002 and in 2004, DALY are available at a regional level according to the WHO's classification (*regional DALY*). In order to cope with data availability, we propose to calculate country Dalys in different ways.

First we can use country DALYs in 2002 or in 2004 (*DALY 2002*; *DALY 2004*) assuming that the figures are representative of the health status over the period under study (Columns 1 and 2 in Table 1). We then use the average country DALY value, calculated with the 2002 and 2004 data (*DALY 2002-2004,* Column 3 in Table 1). Finally, we calculate a *corrected DALY*. Under the hypothesis that the gap between the DALY of a country and the DALY of the WHO region is constant on the 2000-2004's period, the regional DALY is weighted by the ratio of the 2004 country level DALY over the 2004 regional DALY (Column 4 in Table 1). It allows generating DALY at the country level over the whole period and then generates the average value for DALYs. More precisely:

$$Corrected\ Daly\ in\ t = Regional\ DALY\ in\ t \times \frac{Country\ DALY\ in\ 2004}{Regional\ DALY\ in\ 2004}$$ with

$$t = 2000, 2001, 2002, 2004$$

The causes of disease burden differ according to income levels (see footnote 20). In developing countries it is mainly caused by communicable diseases, whereas in rich countries non-communicable diseases are the principal source of disease burden. This characteristic is taken into account while calculating DALYs with respect to communicable diseases and to non-communicable diseases as well. Environmental diseases constitute a non-negligible part of communicable and non-communicable diseases. It is estimated that environmental risk factors contribute to 24% of global burden of disease from all causes (in DALYs), and 23% of of all deaths (Prüss-Üstün and Corvalan, 2011). Finally as malaria and HIV/AIDS constitute respectively a large part of the disease burden in low income countries, and are the fifth main diseases in the world (WHO, 2008), DALYs with respect to both diseases are also considered in the econometric analysis.

We consider several control variables **X**, which are either assumed from the theoretical model or inferred from other cross-country analyses of Solow augmented growth regressions. Initial GDP per capita allows considering conditional convergence when it exhibits a negative effect on growth; annual growth rates of population and investment ratio to GDP have respectively a negative and positive effect on growth (e.g. Mankiw et al. 1992). In addition to the global health indicator, other human capital variables are included. Lagged female school enrollment rates are preferred to male school enrollment as it may also reflect the inequalities that impact growth. Lagged variables may cope with endogeneity bias.

Our second group of control variables includes the Government consumption ratio to GDP, openness and inflation rates, and institutions quality. The government consumption does not have a clear-cut effect on growth (Barro, 1992). For the advocates of bigger government, its

programs provide valuable public goods such as education, health and infrastructure. Government expenditures can also reinforce economic growth by stimulating the demand side of the economy. However, the proponents of smaller government argue that higher spending reduces economic growth by transferring additional resources from the productive sector of the economy to government, which uses them less efficiently. In addition, the expansion of public sector discourages efforts to implement pro-growth policies (tax reform and personal retirement accounts), since the existence of budget deficits can be used as argument to oppose policies that would strengthen the economy (Mitchell, 2005). Openness and inflation allow taking economic policy variables with respectively a positive and a negative effect on growth. Goog institutions are recognized as important determinant of economic performance since property rights and rule of law affect the incentives to invest and innovate (Acemoglu, Johnson and Robinson, 2000). We measure institutional quality using an index of rule of law taken from Kaufmann et al. (2009). This index covers 168 countries, and by construction has a mean of zero and a standard deviation of 1.

Summary statistics are reported in Appendix 4.6 and Appendix 4.7.

4.4.2 Econometric specification

OLS estimation of equation (1) is potentially biased. First there can be a simultaneity bias between global health status and growth (e.g. Bonnel 2000; Bloom, Canning and Malaney 2000; Sachs et al. 1999, 2003; Strauss and Thomas, 2008; Schultz, 2008). Under the hypothesis that faster growing economies have a better health outcome, OLS estimates of health effects on growth are positively biased. Measurement errors of the global health indicator may also induce downward biased estimators (attenuation bias). To deal with these problems, we draw on instrumental variables techniques and therefore identify several instruments.

The first instrument is malaria ecology developed by Kiszewski et al. (2004) and first used in cross-country regressions by Sachs (2003) and Carstensen and Gundlach (2006). Malaria ecology is built upon climatic factors and specific biological properties of each regionally dominant malaria vector which only reflects the forces of biological evolution and is thus independent from present health interventions and economic conditions. Moreover germs likely to be affected by economic conditions or public health interventions (like mosquito abundance, for example) do not enter the calculation of the index (Kiszewski et al. 2004; Carstensen and Gundlach 2006).

The other instrument used in this chapter is the proportion of each country's population threatened by a risk of malaria transmission in 1994 (Sachs 2003). This indicator affects current economic growth only through health status and is unlikely affected by current economic conditions.

4.5. Econometric results

Equation (1) is estimated with the heteroskedastic-efficient two-step generalized method of moments (IV-GMM) estimator which generates efficient coefficients as well as consistent standard errors estimates. The efficiency gains of this estimator relative to the traditional IV/2SLS estimator derive from the use of the optimal weighting matrix, the over-identifying restrictions of the model, and the relaxation of the independently and identically distributed (i.i.d.) assumption. For an exactly-identified model, the efficient GMM and traditional IV/2SLS estimators coincide, and under the assumptions of conditional homoskedasticity and independence, the efficient GMM estimator is the traditional IV/2SLS estimator (Hayashi 2000 pp.206-13 and 226-27; Baum et al. 2007).

4.5.1 Results

The effect of DALYs due to HIV/AIDS on economic development is not estimated for two reasons. First, we did not find a valid and relevant instrument for HIV/AIDS. The instrument used in the literature is the lagged HIV/AIDS variable (McDonald and Roberts, 2006) and we do not have relevant data for that. The second reason is that HIV/AIDS is always associated to co-infections that enter into the group of communicable and non-communicable diseases such as tuberculosis, hepatitis C, liver disease (see for example Sharifi-Mood and Metanat, 2006; Amin et al. 2004). We may thus suppose that the effect of HIV/AIDS may be caught by communicable and non-communicable DALYs.

Besides that restriction, our results stress that health status is an important predictor of economic development on a large sample of poor and rich countries. Efficient-GMM estimations are presented in Table 4.1 below. The quality of the instruments is either validated by the Shea R^2, or the statistic of Fisher and the Hansen over-identification test of the first stage estimation results presented in Appendix 4.9.

The first four columns report estimates with the global DALYs. Contrary to OLS estimates,[21] global health is found to have a negative and statistically significant effect on economic growth thus corroborating the attenuation bias. This result is robust to all the variants of DALYs (country or regional estimations of DALYs, Columns 1 to 4). The marginal effect of the DALY health indicator on growth is significant whatever its calculation (Table 4.1). Contrary to what expected, the coefficient and then the effect of DALYs for communicable diseases (Column 5) are not different from that of the global DALYs. It may

[21] OLS estimates of equation (1) are reported in Appendix 4.8.

reflect the importance of communicable diseases in health status in the world (40% of total DALYs) and as a barrier to economic development. Malaria has however a strong negative effect on economic growth: the coefficient of DALYs for malaria is higher (-0.365) than the coefficients of global DALYs or communicable DALY, also indicating that malaria is one among other health main causes.

Moreover, the other explanatory variables present the expected signs apart from the population growth rate and the education variable. The convergence hypothesis is not rejected, inflation rate reduces economic growth and investment rate improves it. We also find that Government spending is negatively related to economic growth (Landau, 1983). As found in the literature (Knowles and Owen 1994, Berthélemy *et al*. 1997), education is not significant.

Table 4. 1: Two-step GMM estimation of economic effects of DALYs per capita

	Dependent variable: GDP per capita growth						
Independent variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)
DALY in 2002	-0.111**						
	(2.47)						
DALY in 2004		-0.108**					
		(2.55)					
DALY 2002-2004			-0.110**				
			(2.53)				
Corrected DALYs				-0.108***			
				(2.61)			
Communicable DALY					-0.119***		
					(2.64)		
Infectious DALY						-0.157**	
						(2.54)	
Malaria DALY							-0.365**
							(2.36)
Log initial GDP per capita	-0.010***	-0.008**	-0.009**	-0.009***	-0.009**	-0.008**	-0.005*
	(2.61)	(2.49)	(2.57)	(2.59)	(2.56)	(2.44)	(1.74)
Investment ratio to GDP	0.127***	0.105***	0.116***	0.102**	0.110***	0.123***	0.129***
	(3.71)	(2.58)	(3.13)	(2.54)	(2.86)	(3.27)	(3.38)
Population growth rate	0.002	0.002	0.002	0.002	0.003	0.002	0.004*
	(0.55)	(0.87)	(0.71)	(0.82)	(1.31)	(0.60)	(1.77)
Government consumption	-0.108***	-0.119***	-0.114***	-0.117***	-0.117***	-0.111***	-0.134***
	(2.94)	(3.26)	(3.12)	(3.21)	(3.14)	(3.02)	(3.76)
Openness	0.006	0.004	0.005	0.004	0.003	0.005	0.002
	(1.60)	(1.34)	(1.52)	(1.27)	(1.03)	(1.33)	(0.62)
Inflation rate	-0.018**	-0.018**	-0.018**	-0.016**	-0.014*	-0.009	-0.026**
	(2.13)	(2.29)	(2.22)	(2.04)	(1.91)	(1.09)	(2.10)
School enrolment lagged	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000
	(0.97)	(1.21)	(1.09)	(1.18)	(1.06)	(0.35)	(1.25)
Institutions	-0.001	-0.003	-0.002	-0.002	-0.001	-0.000	-0.003
	(0.19)	(0.73)	(0.45)	(0.61)	(0.16)	(0.09)	(0.64)
Constant	0.147***	0.136***	0.141***	0.141***	0.127***	0.111***	0.080***
	(2.93)	(2.92)	(2.95)	(2.99)	(3.00)	(2.91)	(2.65)
Observations	138	138	138	138	138	138	138
R^2	0.345	0.396	0.380	0.393	0.410	0.374	0.411
Shea R2	0.146	0.232	0.190	0.208	0.191	0.157	0.483
Fisher F statistic	6.811	13.726	9.750	11.984	10.924	8.869	54.800
(p-value)	0.0016	0.0000	0.0001	0.0000	0.0000	0.0003	0.0000
Hansen OID p-value	0.467	0.481	0.470	0.624	0.764	0.708	0.274

Note: Health variables are instrumented by Malaria Ecology and Malaria Risk.
***significant at 1%, **significant at 5%, *significant at 10%. Robust t-statistics in parentheses.

4.5.2 Robustness analyses

Our previous results may still be questioned. First, they may be due to the large health outcome gap between developed and developing countries, and may not satisfactorily explain development gaps between developing or developed countries. Secondly, it is relevant to

investigate the role of health in the explanation of development differential within countries which somehow share a common characteristic related to poor basic health infrastructures. Our growth regression is therefore estimated on a low and middle-income countries sub-sample of which results are presented in Table 4.2. First stage estimation results are presented in Appendix 4.10. They are similar to those obtained on the whole sample, namely, health remains an important determinant of economic growth. Coefficients are slightly smaller than those previously obtained on the whole sample (0.083 against 0.108 for Corrected DALYs; 0.324 against 0.365 for Malaria DALYs).

These results suppose that there are other limiting global factors to growth other than health such as education quality which may be not satisfactorily measured.

Table 4. 2: Two-steps GMM estimation of economic effect of DALYs per capita, developing countries

	Dependent variable: GDP per capita growth						
Independent. variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)
DALY in 2002	-0.077**						
	(2.01)						
DALY in 2004		-0.084**					
		(2.07)					
DALY 2002-2004			-0.080**				
			(2.05)				
Corrected DALYs				-0.083**			
				(2.10)			
Communicable DALY					-0.091**		
					(2.05)		
Infectious DALY						-0.108**	
						(2.06)	
Malaria DALY							-0.324*
							(1.88)
Log initial GDP per capita	-0.008**	-0.008*	-0.008*	-0.008**	-0.008*	-0.007*	-0.005
	(1.98)	(1.91)	(1.95)	(1.97)	(1.95)	(1.86)	(1.44)
Investment ratio to GDP	0.128***	0.110**	0.119***	0.109**	0.117***	0.127***	0.124***
	(3.25)	(2.45)	(2.86)	(2.46)	(2.78)	(3.02)	(2.62)
Population growth rate	-0.002	-0.002	-0.002	-0.002	-0.001	-0.001	-0.002
	(0.71)	(0.92)	(0.81)	(0.86)	(0.19)	(0.56)	(0.96)
Government consumption	-0.098***	-0.109***	-0.103***	-0.107***	-0.109***	-0.102***	-0.130***
	(2.66)	(2.90)	(2.80)	(2.86)	(2.85)	(2.76)	(3.21)
Openness	0.001	-0.001	0.000	-0.001	-0.001	-0.000	-0.001
	(0.15)	(0.10)	(0.04)	(0.15)	(0.18)	(0.06)	(0.21)
Inflation rate	-0.021**	-0.021**	-0.021**	-0.019**	-0.018**	-0.015*	-0.028*
	(2.14)	(2.17)	(2.16)	(2.04)	(1.98)	(1.67)	(1.94)
School enrolment lagged	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000
	(0.70)	(0.93)	(0.81)	(0.88)	(0.72)	(0.15)	(1.01)
Institutions	0.002	0.001	0.002	0.001	0.002	0.003	0.002
	(0.52)	(0.18)	(0.36)	(0.21)	(0.45)	(0.60)	(0.35)
Constant	0.130***	0.134***	0.132***	0.137***	0.124***	0.104***	0.102***
	(3.11)	(3.05)	(3.09)	(3.08)	(3.13)	(3.24)	(2.90)
Observations	103	103	103	103	103	103	103
R^2	0.447	0.446	0.452	0.447	0.464	0.468	0.421
SheacR2	0.189	0.265	0.229	0.241	0.211	0.199	0.486
Fisher F statistic	7.748	13.360	10.178	11.784	10.090	9.725	48.174
(p-value)	0.0008	0.0000	0.0001	0.0000	0.0001	0.0002	0.0000
Hansen OID p-value	0.689	0.671	0.679	0.796	0.876	0.862	0.381

Note: Health variables are instrumented by Malaria Ecology and Malaria Risk.
***significant at 1%, **significant at 5%, *significant at 10%. Robust t-statistics in parentheses.

4.5.3 Effect of a standard deviation decrease of the DALYs on growth

In the previous subsection, we showed that population health measured by the global burden of disease has a negative impact on economic development. This result can be

quantified by simulating the effect of a one standard deviation increase of the DALYs on economic growth. The first and third columns of Table 4.3 present respectively the change in economic growth due to one standard deviation decrease of DALYs on the whole sample and that of developing countries. For the total DALYs and communicable diseases DALYs, the effect ranges from 0,44 to 0,50 percentage points on the whole sample and around 0.30 percentage points on the developing countries sample. More importantly, this health impact doubles for infectious diseases and is multiplied by ten for malaria DALYs. The second column of Table 3 shows the average economic growth on the whole sample after experiencing one standard deviation decrease of the DALYs. The average economic growth changes from 4% to around 5.5%, and is even around 10% for malaria DALYs. A similar figure is observed for developing countries sample in the last column. This is largely due to high standard deviation of malaria indicator (around 0.154 against 0.062). One standard deviation decrease of malaria DALYs has the same effect in terms of gain in economic growth on the two samples, while the same decrease in the other sources of DALYs improves more economic growth in the whole sample than in the developing countries sample. This result shows that more efforts must be undertaken on the fight against malaria to enhance economic prosperity in poor countries.

Table 4. 3: Effect of a standard deviation decrease of the global burden of disease on economic growth

	Whole sample		Developing countries sample	
	Change (Δy)	Effect ($y+\Delta y$)	Change (Δy)	Effect ($y+\Delta y$)
DALY in 2002	0,00504	0,04537	0,00297	0,04547
DALY in 2004	0,00455	0,04488	0,00337	0,04587
DALY 2002-2004	0,00473	0,04507	0,00315	0,04565
Corrected DALYs	0,00442	0,04476	0,00324	0,04574
Communicable DALY	0,00534	0,04568	0,00405	0,04655
Infectious DALY	0,00976	0,05010	0,00566	0,04816
Malaria DALY	0,05630	0,09663	0,05609	0,09859

4.6. Concluding remarks

This chapter deeply assesses the effect of health on economic growth. It contributes to the debate on the relationship between health outcomes and economic performance by paying a particular attention to global health status measurement issues. Using the Disability-Adjusted Life Year proposed by the World Bank and World Health Organization in 1993, we argue that, traditional health indicators such as life expectancy and mortality rates are not relevant proxies of health status in a population. They present many drawbacks and are devoted to a particular health problem, and they do not measure the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability.

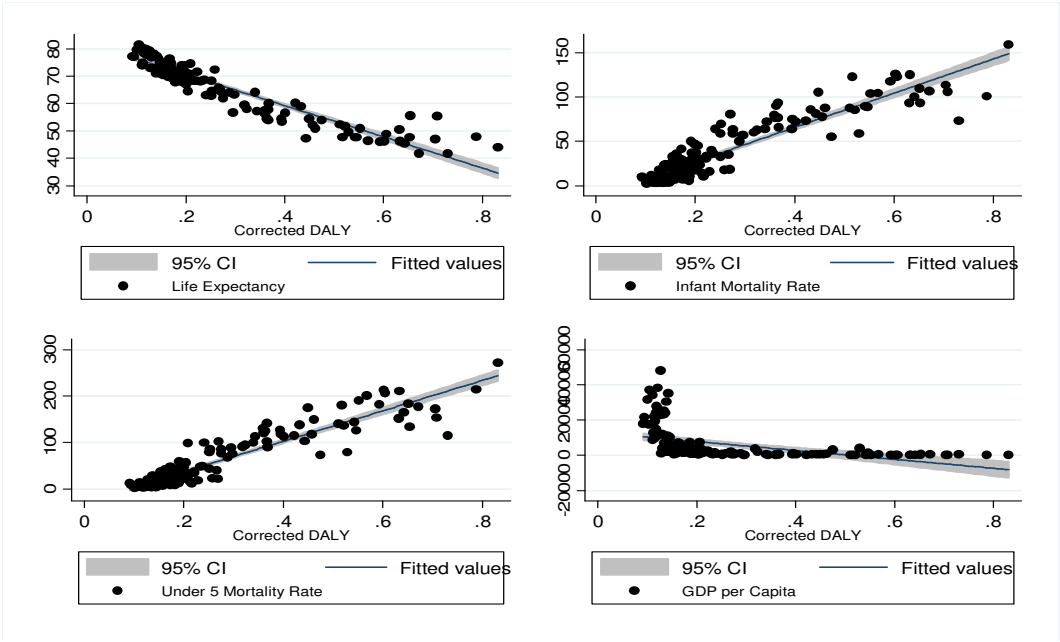
Several remarks can be drawn from our results. First, as the results were very similar whatever the estimation of DALYs used (corrected DALYs, country DALYs or regional DALYs), it appears that regional DALYs can be considered as good proxies of the disease burden of each country within a region. Estimating country DALYs each year does not seem to be necessary. Secondly we highlight and confirm the role of poor health in the economic development. This result has been established using a global health outcome which takes into consideration mortality, morbidity, and disability consequences of health as well. Thus, we estimated the effect of global health, and not only the effect of specific diseases or fatal diseases consequences. However, this indicator that can be calculated for a group of particular diseases such as communicable diseases, or for a specific disease, such as malaria, allow us to estimate the economic burden of diseases that remain an important impediment to economic development especially in low income countries.

These results call for important and relevant policy recommendations, especially for the developing world.

For this challenge to be transformed into an opportunity, accurate health policies should be implemented, such as efficient health spending. More attention should be paid to water and sanitation that are the main determinants of communicable diseases such as diarrheal diseases. International community should also help national health policy makers through their support and pressure. This could be done through increasing health sector assistance and the promotion of good institutions. Brain drain in health sector also should be transformed into brain gain through support to physicians from poor countries.

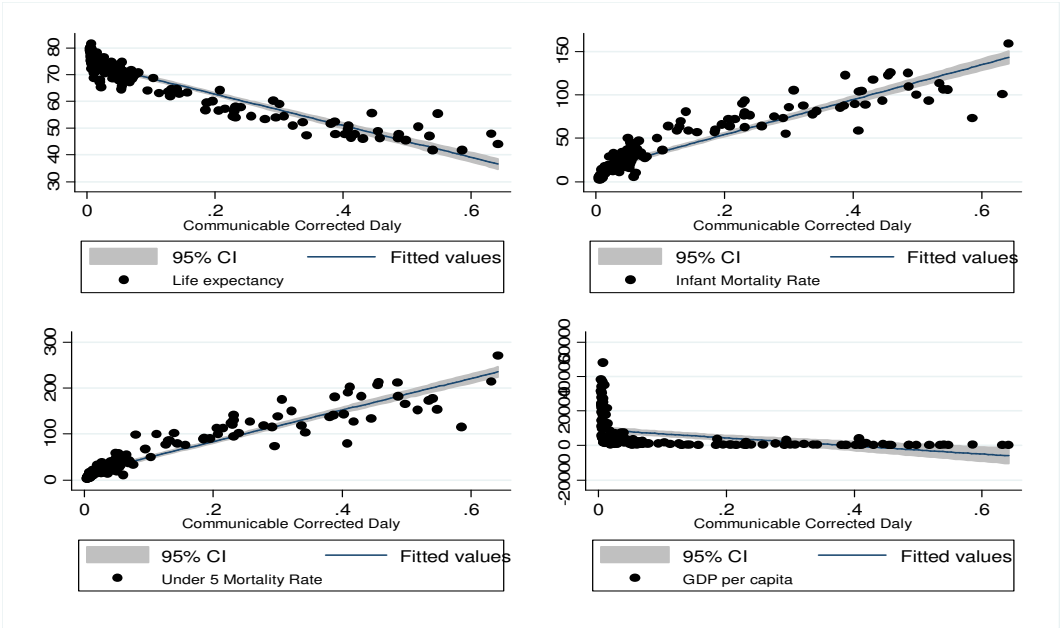
Appendices 4

Appendix 4.1: Relationship between Corrected DALY, traditional health indicators and GDP per capita.



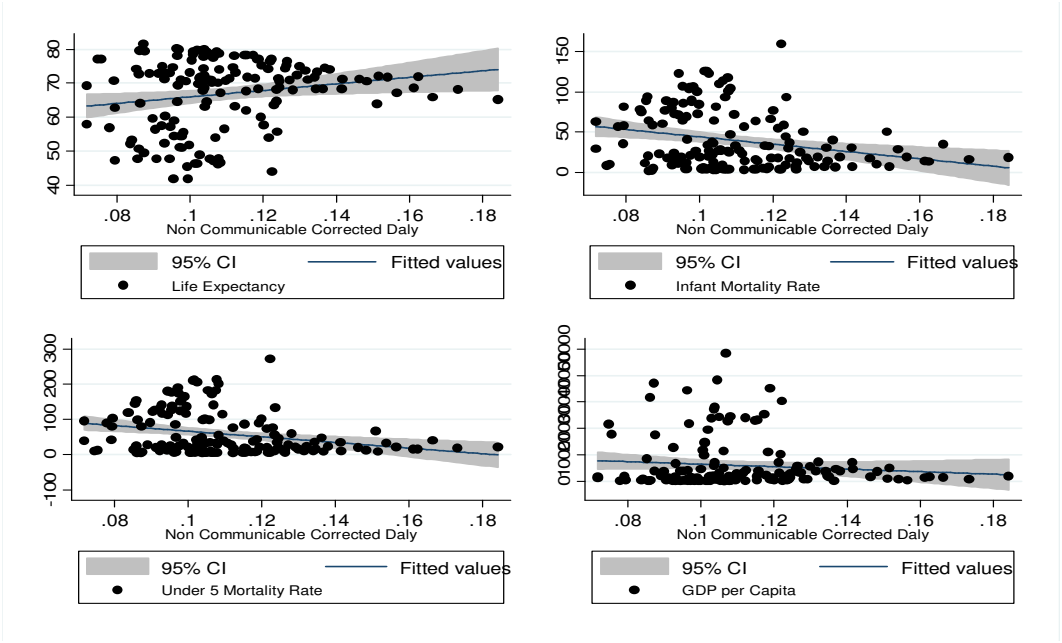
Source: Authors' construction with data from World Bank and WHO.

Appendix 4.2: Relationship between Communicable Corrected DALY, traditional health indicators and GDP per capita.



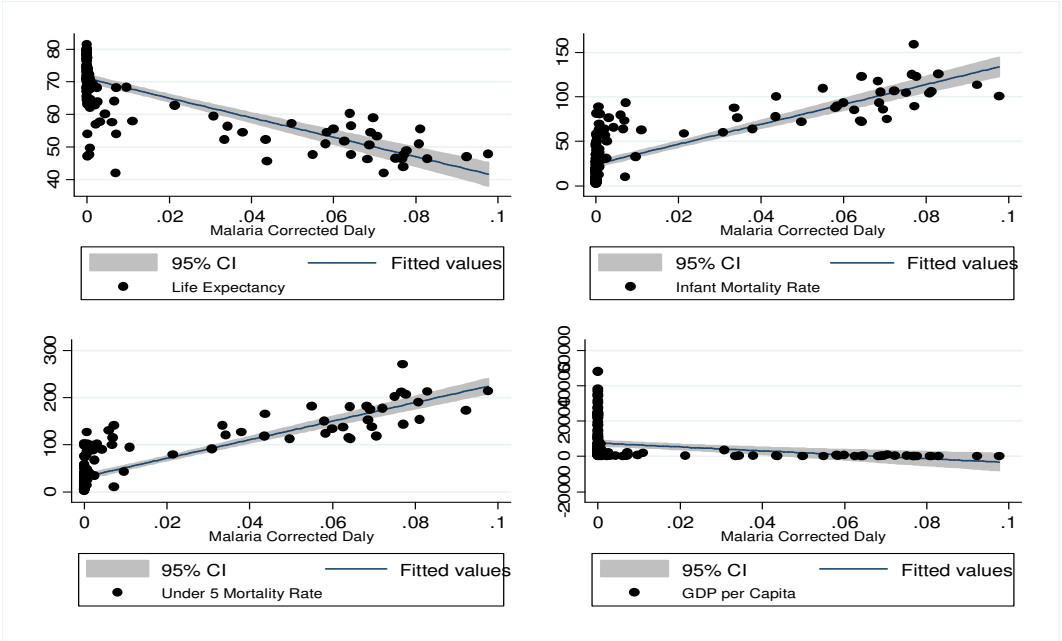
Source: Authors' construction with data from World Bank and WHO.

Appendix 4.3: Relationship between Non Communicable Corrected DALY, traditional health indicators and GDP per capita.



Source: Author's construction with data from World Bank and WHO.

Appendix 4.4: Relationship between Malaria Corrected DALY, traditional health indicators and GDP per capita.



Source: Authors' construction with data from World Bank and WHO.

Appendix 4.5: Literature review on the effect of health on economic growth

Study	Health indicator	Coefficient (standard error)	data	Estimator	Other covariates
Barro and Lee (1994)	Life expectancy	0.073 (0.013)	Two periods n=85 for 1965–75, n=95 for 1975–85	SUR with country random effects	Male and female secondary schooling, I/GDP, G/GDP, log(1+black market premium), revolutions
Cuddington and Hancock (1994)	AIDS	0.2- 0.3% points lost in the medium case and 1.2-1.5 in the lower case	Each five year age cohort from 1985-2010 in Malawi	simulation	Na
Barro and Sala IMartin (1995)	Life expectancy	0.058 (0.013)	Two periods n=87 for 1965–75, n=97 for 1975–85	SUR with country random effects	Male and female secondary and higher education, log(GDP) · human capital, public spending on education/ GDP, investment/GDP, government consumption/GDP, log(1+black market premium), political instability, growth rate in terms of trade
Barro (1996)	Life expectancy	0.042 (0.014)	Three periods 1965–75, n=80; 1975–85, n=87; 1985–90, N=84	3SLS using lagged values of some regressions as instruments, period random effects	Male secondary and higher schooling, log(GDP) · male schooling, log fertility rate, government consumption ratio, rule of law index, terms of trade change, democracy index, demo- cracy index squared, inflation rate, continental dummies
Caselli, Esquivel, and Lefort (1996)	Life expectancy	-0.001 (0.032)	25-year panel at 5-year intervals, 1960–85, n=91	GMM (Arellano- Bond method)	Male and female schooling, I/GDP, G/GDP, black market premium, revolutions
Sachs and Warner (1997)	Life expectancy	45.48 (2.60)	25-year cross-section, N=79	OLS	Openness, openness xlog(GDP), land-locked, government saving, tropical climate, institutional quality, natural resource exports, growth in economically active population minus population growth
	life expectancy squared	-5.40 (2.41)			

Study	Health indicator	Coefficient (standard error)	data	Estimator	Other covariates
Bloom and Sachs (1998)	Life expectancy	0.037 (0.011)	25-year cross-section, 1965–90, n=65	OLS	Log secondary schooling, openness, institutional quality, central government deficit, percentage area in tropics, log coastal population density, log inland population density, total population growth rate, working- age population growth rate, Africa dummy
Bloom and Malaney (1998)	Life expectancy	0.027 (0.107)	25-year cross-section, 1965–90, n=77	OLS	Population growth, growth of economically active populations, log years of secondary schooling, natural resource abundance, openness, institutional quality, access to ports, average government savings, tropics, ratio of coastline distance to land area
Bloom and Williamson (1998)	Life expectancy	0.040 (0.010)	25-year cross-section, 1965–90, n=78	OLS	Population growth rate, working-age population growth rate, log years of secondary schooling, natural resource abundance, openness, institutional quality, access to port, average government savings rate, tropics dummy, ratio of coastline to land area
Gallup, Sachs. and Mellinger (1999)	life expectancy	2.4 (1.34)	25-year cross-section, 1965–90, n=75	2SLS with malaria index instrument by temperate (temperate, boreal, and polar eco-zones), desert (tropical and subtropical deserts), subtropical (non desert subtropical), and tropical (non desert tropical)	Years of secondary schooling, openness, quality of public institutions, population within 100 kilometers of the coast, malaria index in 1966, change in malaria index from 1966 to 1994
	Malaria index 1966	-2.6 (0.67)			
Hamoudi and Sachs (1999)	Life expectancy	0.072 (0.020)	15-year cross-section, 1980–95, n=78	OLS	Institutional quality, openness, net government savings, tropics land area, log coastal population density, population growth rate, working-age population growth rate, Africa dummy
	Infant mortality rate	-0.0002 (0.00008)			

Study	Health indicator	Coefficient (standard error)	data	Estimator	Other covariates
Bloom, Canning and Malaney (2000)	Life expectancy	0.019 (0.012)	25-year cross section, 1965–90, n=80	2SLS	Log of ratio of total population to working-age population, tropics, log of years of secondary schooling, openness, institutional quality, population growth rate, working age population growth rate
Bonnel (2000)	HIV/AIDS	-0.7% points per year	1990- 1997 African countries	OLS and 2SLS	Log GDP 1990, Log phone per capita, Macro rating, Law rating, Primary enrollment rate, Malaria morbidity and dummy
Ranis and Steward (2000)	Life expectancy	0.06 (0.016)	N=73 developing countries Cross country 1960-1992	2SLS using lagged values as instruments	change in the log of life expectancy 1962-82, gross domestic investment, income distribution, regional dummies,
Bhargava, Jamison, Lau, and Murray (2001)	Adult survival rate	0.358 (0.114)	25-year panel at 5-year intervals, 1965–90, n= 92	Dynamic random effects	Tropics, openness, log fertility, log (Investment/GDP)
	ASR xlog (GDPC)	-0.048 (0.016)			
Mayer (2001)	Probability of survival by age and gender groups	0,8 and 1,5%	Panel of 18 countries over 1975, 1980 and 1985	Granger-type causality tests	Schooling, investment, Government consumption and fertility
Gallup and Sachs (2001)	falciparum malaria index	-2.5 (0.71)	25-year cross-section, 1965–90, n=75	2SLS with the prevalence of 53 different Anopheles mosquito vectors in each country in 1952 as instrument	Years of secondary schooling, openness, quality of public institutions, population within 100 kilometers of the coast, malaria index in 1966, change in malaria index from 1966 to 1994
	life expectancy	3.0 (0.84)			
Arora (2001)	Stature at Adulthood, Life Expectancy	30- 40%	10 developed countries over the course of 100 to 125 years	Cointegration and Error-Correction	Na
Sachs (2003)	Malaria Risk	-1.43 (0.35)	Cross- country regression in 1995, N=101	2SLS with Malaria Ecology as instrument	rules of law
Bloom, Canning and Sevilla (2004)	Life expectancy	0.040 (0.019)	every 10 years from 1960 to 1990	Nonlinear two stage least squares with lagged as instrument	Capital, labor, Schooling, Experience, Technological catch-up coefficient, Percentage of land area in the tropics and Governance

Study	Health indicator	Coefficient (standard error)	data	Estimator	Other covariates
Aguayo-Rico, and Guerra-Turrubiates (2005)	Health services,	0,0021 (0.006)	N=104 panel 1970, 1980 and 1990	OLS	capital, labor, schooling, Environment
	Lifestyles	0.0016 (0.0002)			
	total health index	0.0015 (0.0001)			
Bloom and Canning (2005)	Adult survival rate	0.03 (0.009)	5 years panel from 1960 to 1995	OLS	capital, labor, schooling, Environment, Technological catch-up coefficient, Percentage of land area in the tropics, Openness, Percentage oft land within 100 kilometers of the coast, Ethno-linguistic fractionalization, Institutional quality
Lorentzen, McMillan and Wacziarg (2005)°	adult mortality rate	-8.564 (2.23)	cross-country 1960-2000	2SLS with malaria ecology, climatic factors and geographic characteristic as instruments	investment, education, Government consumption, openness, population, interstate battle death
	crude death rate	-145.765 (64.78)			
	infant mortality rate	-31.644 (6.92)			
Acemoglu and Johnson (2006)	Life expectancy	-1.43 (2.24)	Panel 1940-1980, N=234 and 47 countries	2SLS with predicted mortality, as instrument	Population, investment, education
Carstensen and Gundlach (2006)	Malaria risk	-1.31 (0.42)	Cross country of 45 countries	2SLS with malaria ecology as instrument	Institutional quality, climatic factors and geographic characteristic
McDonald and Roberts (2006)	HIV/AIDS	-0.59%	Panel of each five year from 1960 to 1998 for all countries.	2SLS with lagged as instruments and GMM	Income per capita, investment, population growth, schooling, proteins, malaria, infant mortality, life expectancy.
Weil (2007)	height, adult survival rates, and age at menarche	9.9-12.3% income variation explained by health	cross- country regression in 1996, N=73	2SLS with health inputs as instruments	investment, education

Appendix 4.6: Variables characteristics and sources

	mean	min	max	Coef of Var.	Std error	Source
GDP. growth	0,04	-0,06	0,13	0,65	0,03	WDI
Corrected DALYs	0,27	0,10	0,83	0,65	0,17	WHO
DALY 2002-2004	0,27	0,10	0,89	0,66	0,18	WHO
DALY in 2002	0,28	0,10	0,95	0,68	0,19	WHO
DALY in 2004	0,26	0,10	0,82	0,64	0,17	WHO
Communicable DALY	0,13	0,004	0,64	1,30	0,17	WHO
Infectious DALY	0,08	0,001	0,56	1,47	0,12	WHO
Malaria DALY	0,01	0,00	0,09	1,95	0,02	WHO
Malaria Ecology	3,86	0,00	31,55	1,77	6,85	Sachs 2003
Malaria Risk	0,37	0,00	1,00	1,18	0,44	Sachs 2003
Investment ratio to GDP	0,21	0,08	0,57	0,33	0,07	WDI
Population growth rate	1,38	-1,10	7,07	0,86	1,20	WDI
Government consumption	0,16	0,05	0,53	0,40	0,07	WDI
Openness	0,86	0,22	2,68	0,48	0,42	WDI
Inflation rate	0,10	-0,01	2,03	2,36	0,23	WDI
School enrollment	100,77	36,53	144,52	0,17	16,75	WDI
rule of law	-0,05	-1,90	2,01	-19,93	0,96	Kaufmann Kraay

Appendix 4.7: Correlation between the main variables

	GDP Growth	Corrected DALYs	DALY 2002-2004	DALY in 2002	DALY in 2004	Communicable DALY	Infectious DALY
Corrected DALYs	0,005	1,00					
DALY 2002-2004	0,03	0,99*	1,00				
DALY in 2002	0,03	0,97*	0,99*	1,00			
DALY in 2004	0,03	1,00*	0,99*	0,97*	1,00		
Commun. DALY	-0,02	0,99*	0,98*	0,97*	0,98*	1,00	
Infectious DALY	-0,08	0,95*	0,96*	0,95*	0,94*	0,97*	1,00
Malaria DALY	0,03	0,84*	0,83*	0,80*	0,84*	0,84*	0,78*

Appendix 4.8: OLS estimation of the economic effects of health status

	Dependent variable: GDP per capita growth						
Independent. variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)
DALY in 2002	-0.013						
	(0.49)						
DALY in 2004		-0.019					
		(0.60)					
DALY 02-04			-0.016				
			(0.54)				
Corrected DALYs				-0.023			
				(0.81)			
Communicable DALY					-0.034		
					(1.20)		
Infectious DALY						-0.044	
						(1.43)	
Malaria DALY							-0.183
							(1.62)
Log initial GDP per capita	-0.004	-0.004	-0.004	-0.005*	-0.005**	-0.005*	-0.005*
	(1.55)	(1.57)	(1.56)	(1.72)	(1.99)	(1.98)	(1.85)
Investment ratio to GDP	0.119***	0.116***	0.118***	0.115***	0.114***	0.117***	0.116***
	(3.71)	(3.64)	(3.69)	(3.61)	(3.59)	(3.62)	(3.54)
Population growth rate	0.005*	0.005**	0.005**	0.005**	0.006**	0.005*	0.006**
	(1.96)	(2.02)	(1.99)	(2.01)	(2.16)	(1.93)	(2.36)
Government consumption	-0.088***	-0.089***	-0.089***	-0.089***	-0.091***	-0.091***	-0.100***
	(2.84)	(2.81)	(2.82)	(2.81)	(2.80)	(2.86)	(2.97)
Openness	0.004	0.004	0.004	0.004	0.004	0.004	0.003
	(1.26)	(1.19)	(1.23)	(1.17)	(1.07)	(1.14)	(0.93)
Inflation rate	-0.025**	-0.025**	-0.025**	-0.024**	-0.023**	-0.021**	-0.026**
	(2.46)	(2.40)	(2.43)	(2.36)	(2.36)	(2.34)	(2.31)
School enrolment lag	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000
	(0.28)	(0.34)	(0.31)	(0.40)	(0.45)	(0.28)	(0.62)
Institutions	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004
	(1.19)	(1.21)	(1.19)	(1.18)	(1.02)	(1.05)	(0.98)
Constant	0.051*	0.055*	0.053*	0.060**	0.064***	0.058***	0.060***
	(1.76)	(1.82)	(1.77)	(2.10)	(2.65)	(2.83)	(2.62)
Regional dummies	yes	yes	yes	yes	yes	yes	yes
Observations	153	153	153	153	153	153	153
R^2	0.378	0.380	0.379	0.382	0.389	0.391	0.388

Note: ***significant at 1%, **significant at 5%, *significant at 10%. Robust t-statistics in parentheses.

Appendix 4.9: first stage estimation results (whole sample) ++

	(1) DALY 2002	(2) DALY 2004	(3) DALY 02-04	(4) Corr. DALY	(5) Comm. DALY	(6) Infect. DALY	(7) Mal. DALY
Malaria Ecology	0.006**	0.006**	0.006**	0.005*	0.004	0.003*	0.002***
	(2.16)	(2.43)	(2.36)	(1.97)	(1.64)	(1.77)	(5.54)
Malaria Risk	0.084*	0.087**	0.085**	0.102**	0.104***	0.075**	0.015***
	(1.82)	(2.44)	(2.13)	(2.56)	(2.76)	(2.33)	(2.96)
Regional dummies	yes	yes	yes	yes	yes	yes	yes
Observations	138	138	138	138	138	138	138
Fisher F-Stat.	6.81	13.72	9.75	11.98	10.92	8.86	54.80
Shea partial R^2	0.14	0.23	0.19	0.20	0.19	0.15	0.48
Hansen OID p-val.	0.46	0.48	0.47	0.62	0.76	0.70	0.27

Note: ***significant at 1%; **significant at 5%; *significant at 10%. t-statistics in parentheses.
++ We show only the coefficients of the instruments, but all the exogenous variables are included in the regressions

Appendix 4.10: first stage estimation results (Developing countries) ++

	(3) DALY 2002	(4) DALY 2004	(2) DALY 02-04	(1) Corr. DALY	(5) Comm. DALY	(6) Infect. DALY	(7) Mal. DALY
Malaria Ecology	0.006**	0.006**	0.006**	0.005**	0.004*	0.004*	0.002***
	(2.40)	(2.61)	(2.58)	(2.11)	(1.75)	(1.95)	(5.36)
Malaria Risk	0.123**	0.110***	0.117***	0.128***	0.125***	0.104***	0.015**
	(2.44)	(2.97)	(2.72)	(3.14)	(3.08)	(2.90)	(2.50)
Regional dummies	yes	yes	yes	yes	yes	yes	yes
Observations	103	103	103	103	103	103	103
Fisher F-Stat.	7.74	13.36	10.17	11.78	10.09	9.72	48.17
Shea partial R^2	0.19	0.26	0.23	0.24	0.21	0.20	0.48
Hansen OID p-val.	0.69	0.67	0.68	0.79	0.87	0.86	0.38

Note: ***significant at 1%; **significant at 5%; *significant at 10%. t-statistics in parentheses.
++ We show only the coefficients of the instruments, but all the exogenous variables are included in the regressions

Appendix 4.11: List of countries

Low-income	**Lower-middle-income**	**Upper-middle-income**	**High-income**
Benin	Albania	Argentina	Bahrain
Burkina Faso	Algeria	Belarus	Estonia
Burundi	Armenia	Belize	Israel
Cambodia	Azerbaijan	Botswana	Kuwait
Central African Republic	Bhutan	Brazil	Malta
Chad	Bolivia	Bulgaria	Oman
Comoros	Cameroon	Chile	Slovenia
Congo, Dem. Rep.	Cape Verde	Costa Rica	Trinidad and Tobago
Cote d'Ivoire	China	Croatia	United Arab Emirates
Eritrea	Colombia	Dominica	Australia
Ethiopia	Congo, Rep.	Fiji	Austria
Gambia, The	Djibouti	Gabon	Belgium
Ghana	Dominican Republic	Grenada	Canada
Guinea	Ecuador	Jamaica	Czech Republic
Guinea-Bissau	Egypt, Arab Rep.	Kazakhstan	Denmark
Kenya	El Salvador	Latvia	Finland
Kyrgyz Republic	Georgia	Libya	France
Liberia	Guatemala	Lithuania	Germany
Madagascar	Guyana	Malaysia	Greece
Malawi	Honduras	Mauritius	Hungary
Mali	India	Mexico	Iceland
Mauritania	Indonesia	Panama	Ireland
Mozambique	Iran, Islamic Rep.	Poland	Italy
Nepal	Jordan	Romania	Japan
Niger	Lesotho	Russian Federation	Korea, Rep.
Nigeria	Macedonia, FYR	Seychelles	Luxembourg
Pakistan	Maldives	South Africa	Netherlands
Rwanda	Moldova	St. Kitts and Nevis	New Zealand
Sao Tome and Principe	Mongolia	St. Lucia	Norway
Senegal	Morocco	St. Vincent and the Grenadines	Portugal
Sierra Leone	Namibia	Suriname	Slovak Republic
Tajikistan	Nicaragua	Turkey	Spain
Tanzania	Paraguay	Uruguay	Sweden
Togo	Peru	Venezuela, RB	Switzerland
Uganda	Philippines		United Kingdom
Uzbekistan	Sri Lanka		United States
Vietnam	Sudan		
Yemen, Rep.	Swaziland		
Zambia	Syrian Arab Republic		
Zimbabwe	Thailand		
	Tonga		
	Tunisia		
	Ukraine		

Chapter 5: Interrelationships between environment quality, health and economic activity: What consequences for economic convergence?[22]

[22] A version of this chapter was published under the reference: Drabo, A., 2010. "Environment Quality and Economic Convergence: Extending Environmental Kuznets Curve Hypothesis." ***Economics Bulletin***, 30(2), 1617-1632.

5.1. Introduction

Environmental protection is an important issue that is gradually more present in the development strategies. It occupies a significant place in economic policies and constitutes a major concern for the international community. This concern expressed at international level, is illustrated in many international meetings and conferences: two Nobel Peace Prizes were awarded to the personalities who raised public awareness on environmental issue (Wangari Maathai 2004 and Al Gore 2007), it is at the of the creation of the Intergovernmental panel on Climate Change (IPCC), and it is one of the eight Millennium Development Goals (MDGs) adopted by the United Nations in 2000. In fact, 192 United Nations member states undertook in 2000 to "integrate the principles of sustainable development into country policies and programmes; reverse loss of environmental resources; reduce biodiversity loss and halve, by 2015, the proportion of people without sustainable access to safe drinking water and basic sanitation." This great interest is explained by the fact that environment is intimately connected to a viable ecosystem as explained by the United Nations Secretary General in the United Nations Environmental Programme (UNEP) 2007 annual Report: "it keeps the climate stable, clothes our backs, provides the medicines we need and protects us from radiation from space."

Although environmental protection is nowadays an important emerging concept, the search for a large and sustainable pro poor economic growth remains a necessity and a priority for all economies. The simultaneous pursuit of these two objectives, gives rise to the question of what is the relationship between economic activity and environmental degradation. During the early decades, many authors tried to give theoretical and empirical responses to this question and the most popular remains the Environmental Kuznets Curve Hypothesis (EKC). The EKC (Grossman 1995; Grossman and Krueger 1995; Torras and Boyce 1998) describes the relationship between declining environmental quality and income as an inverted-U, that is, in

the course of economic growth and development, environmental quality initially worsens but ultimately increases with improvements in income levels.

The relationship between income and environmental quality should not be limited to the EKC, since the environmental degradation in turn can have significant effects on economic activity (Bovenberg and Smulders 1995 and 1996; Bruvoll et al. 1999). These effects impact growth through many channels among which health status. Indeed, environmental degradation reduces the availability labour force, and decrease the productivity of those who are working because of the health problem it provokes. Some works estimate the cost of pollution, and they show that morbidity and mortality should be considered (Scapecchi 2008).

This interrelationship between environment, health, and economic activity can have different consequences depending on the development level and this can slow down the speed of economic convergence. In fact, given the environmental Kuznets Curve hypothesis, in the early stage of economic development, the gain from income growth could be cancelled or mitigated by environmental degradation through populations' health (and other channels) and create a vicious circle in economic activity unlike in developed countries. This in turn could slow down economic convergence.

The aim of this chapter is to assess i) the impact of environmental degradation on economic convergence, and ii) the role played by the relationship among environmental quality, health, and economic activity on the modification of the speed of convergence due to environmental variation.

The interest of this chapter comes from the fact that very few studies are interested, in a simultaneous way, in these three elements (or pillars) in spite of the importance granted by the international community. The major part of international studies on this relation, nevertheless, focuses on the EKC hypothesis and those interested in the reverse causality are mainly

theoretical works. Moreover, from our knowledge this is the first work investigating the association between economic convergence, environmental degradation, and health.

Our study shows that there is a feedback relationship between economic activity and environmental quality on one hand and between health and economic activity on the other hand. Health status remains an important channel through which environmental degradation affects economic growth even if it is not the only one. Environmental degradation affects negatively economic activity and reduces the ability of poor countries to reach developed ones economically.

The remaining of this chapter is organised in five sections. Section 2 explains through a theoretical model, the impact of environment quality on economic convergence. Section 3 reviews the literature on the relationship between economic activity, environment, and health. Section 4 is devoted to the empirical design. In this section, we investigate the association between environmental indicators and economic convergence before examining the relationship between environmental degradation, health, and economic growth through an econometric technique. Section 5 presents the results and section 6 concludes.

5.2. Literature review

The review of the literature concerns the link between economic growth and environmental quality, and then the effect of environmental degradation, namely pollution, on population's health.

5.2.1 Economic growth and environment

Growth and economic convergence

Solow (1956) growth model has been tested and improved by economists. It was generalized by Barro and Sala-i-Martin (1992), Mankiw, Romer and Weil (1992), Levine and Renelt (1992) with the conditional convergence. Conditional convergence implies that countries will reach their own steady states. Hence, when looking for convergence in a cross country study, it is necessary to control for differences in steady states of different countries. The choice of control variables is crucial because the statistical significant level as well as the coefficient amplitude of the variable of interest is sensitive to this choice (Levine and Renelt 1992). Mankiw, Romer and Weil (1992) provided an analysis of economic convergence by adding human capital, represented by education level, to Solow's model (1956) and they showed that their results fit better to the predictions of Solow theoretical model. Knowles and Owen (1994) completed this work by adding health as second human capital variable.

All these improvements are important but not enough because they do not take into account the role that could play some omitted variables, in particular the environmental quality which arouses a renewed interest these last years with the natural resources curse and EKC hypothesis.

Consideration of the environmental aspect

The existence of an intrinsic relation between economic activity and environmental quality remains evident. At the theoretical level several authors tried to give an explanation to the way the environment degradation could impact economic activity (Bovenberg and Smulders 1995 and 1996; Bruvoll et al. 1999; Resosudarmo and Thorbecke 1996; Hofkes 1996; Geldrop and Withagen 2000). These theoretical works can be divided into four major categories following Panayotou (2000) (See Appendix 5.1). Optimal growth models built on a

Ramsey (1928) model, as extended by Koopmans (1960) and Cass (1965), constitute the first category (Keeler et al. 1971; Mäler 1974; Gruver 1976; Brock 1977; Becker 1982; Tahvonen and Kuuluvainen 1994; Selden and Song 1995 and Stokey 1998). Some of these models considered the effects of pollution on growth path (Keeler et al. 1971; Gruver 1976, Van der Ploeg and Withagen 1991) whereas others focused on natural resources depletion (Dasgupta and Heal 1974; Solow 1974). In general, models of pollution and optimal growth suggest that some abatement or curtailment of growth will be optimal.

The second category considers not only pollution as an argument of production and utility functions, but also it includes environment itself as a factor of production (Lopez 1994; Chichilnisky 1994; Geldrop and Withagen 2000). This measure of environmental quality can be conceptualized as a stock that is damaged by production or pollution. The presence of environmental stock in the production function means that optimal pollution taxes or regulations are not sufficient to achieve the optimal level of environmental quality in the steady state.

The third group is constituted of endogenous growth models that relax the neoclassical specification of the production function assumed in the optimal growth models (Bovenberg and Smulders 1995 and 1996; Hofkes 1996; Ligthhard and Van der Ploeg 1994; Gradus and Smulders 1993, and Stokey 1998). Based on the works of Romer (1986, 1990), these models are characterised by constant or increasing returns to scale to some factors, or a class of factors, because private returns on investment may differ from the social returns on investment, often because of externality effects. This category consists in extending this new growth theory to include the environment or pollution as factor of production and environment quality as an argument of the utility function. Bovenberg and Smulders (1995, 1996) modify the Romer (1986) model to include the environment as a factor of production. Lighhard and Van der Ploeg (1994), Gradus and Smulders (1993) and Stockey (1998) extend

the simple “AK” used by Barro by including environment. Hung, Chang and Blackburn (1994) use the Romer (1990) work. In general, optimal pollution control requires a lower level of growth than would be achieved in the absence of pollution.

Finally, we have other models that connect environmental degradation and economic growth. This category includes the overlapping generation model based on Diamond (1965), it is the case of John and Pecchenino (1994, 1995). We also have a two country general equilibrium model of growth and environment in presence of trade (Copeland and Taylor 1994). These models reinforce the results of the optimal growth models.

At the empirical level, the effect of environment on growth is studied by two papers through the different channels such as labour supply, labour productivity, and physical capital, using simulation Models. Bruvol et al. (1999) estimated the cost to society of environmental constraints, called environmental drag, in Norwegian economy through a dynamic resource environment applied model (DREAM). Their simulation indicates that the environmental drag reduces annual economic growth rate by about 0,82 percentage points because of the fall in labour supply and capital between 1988 and 2030. Indeed, the corrosion from pollution is estimated to increase the depreciation rate of public and private investment, and the depreciation of public capital increases public consumption, which crowds out private consumption. In addition, their results show that, with an increase of 28%, health damages from emissions are the most important compared to other damage factors, and contribute to 39% of the total disutility. The other main cost components to development are congestion and traffic accidents.

Moreover, Resosudarmo and Thorbecke (1996) show through the Social Environmental Accounting Matrix (SEAM) and some simulations with Indonesian data, that the improvement of environment quality by the “Blue Sky Program” reduces health problems, and therefore stimulates economic growth.

Effect of economic growth on environment: The EKC hypothesis

We have shown that environmental quality affects economic performance. Economic activity in turn may deteriorate environment quality (Shafik 1994, Mansour 2004; Yadav 1997; WRI 1996; Hettige, Mani and Wheeler 1998). During the 1990's, scholars have investigated theoretically and empirically the effect of economic development on pollution, and the most popular finding remains the Environmental Kuznets Curve Hypothesis (EKC). The EKC (Grossman 1995; Grossman and Krueger 1995; Torras and Boyce 1998) describes the relationship between declining environmental quality and income as an inverted-U, that is, in the course of economic growth and development, environmental quality initially worsens but ultimately improves with improvements in income level. The first explanation for the EKC relationship is that the environment can be thought of as a luxury good. In the early stage of economic development a country would be unwilling to exchange consumption for investment in environmental regulation, hence environmental quality declines. When the country reaches the threshold level of income, its citizens start to demand improvement in environmental quality. Another explanation of the EKC hypothesis is that countries pass through technological life cycles, as they move from high polluting technology (agriculture-based economies) to less polluting technology (service-based systems). In addition to these macroeconomic explanations, the EKC hypothesis is supported by some microeconomic foundations (Andreoni and Levinson 2001).

5.2.2 Environment and health

Healthy population is essential for the development of an economy and requires a healthy environment (clean air, water, recreation and wilderness). As argued by Pearce & Warford (1993), the immediate and most important consequences of environmental degradation are

damages to human health through different forms of diseases. Many authors have assessed how air quality may be associated to population's health. Some scholars showed that air pollutions increase mortality rate (Woodruff et al., 1997 ; Gangadharan & Valenzuela, 2001; Chay et al. 2003; Aunan & Pan, 2004; Jerrett et al., 2005).

Other authors assess the link between pollution and particular illness, such as cardio-respiratory disease (Aunan & Pan, 2004; Burnett & Krewski, 1994; Jerrett et al., 2005), asthma (Nauenberg & Basu, 1999) and congenital anomalies (Rankin et al., 2009).

There is therefore a link between environmental quality, people health and economic performance. This interrelationship provokes different consequences depending on development level if the EKC hypothesis is verified. In countries below EKC income threshold, all attempts to boost economic growth (without abatement) will result in greater environmental degradation. And this will burden economic growth through health and other channels creating a vicious circle. However, when countries above the EKC income threshold try to boost their economic growth, their environment quality will be improved and therefore they will be in a virtuous circle. That will penalize poor countries by slowing down the speed of convergence if they do not take care of environmental concern.

5.3. Environmental quality and economic convergence: The model

The object of this model is to theoretically investigate the association between environmental quality and economic convergence. We introduce environment variable in an augmented Solow growth model, and we observe the consequences on economic convergence process. In this model, unlike Mankiw, Romer and Weil (1992), environmental capital and human capital are treated as labour augmenting rather than entering the production function as separate factors of production (Knowles and Owen, 1997).

We begin this model by a neoclassical growth model.

$$Y_{it} = K_{it}^{\alpha} \hat{L}_{it}^{1-\alpha} \tag{3.10}$$

Where *Y* is the real output, *K* is the stock of physical capital, α is the capital share, 1-α is the labour share, and

$$\hat{L}_{it} = A_{it} Q_{it}^{\theta 1} H_{it}^{\theta 2} L_{it} \tag{3.11}$$

L is the raw labour input, *A* the technological progress, *Q* is the natural environment quality and *H* is the measure of human capital. $\hat{L}$ represents an effective labour input. $\theta 1$ and $\theta 2$ represent the labour augmenting elasticities of environment and human capital.

The equation (3.10) can be written in per unit of effective labour:

$$\hat{y}_{it} = k_{it}^{\alpha} \tag{3.12}$$

With $\hat{y}_{it} = Y_{it} / \hat{L}_{it}$ and $k_{it} = K_{it} / \hat{L}_{it}$. We assume that *L*, *Q*, *H* and A grow at constant rate *n*, *q*, *h* and *g* respectively.

The accumulation of physical capital can be modelled as (3.13).

$$\dot{k}_{it} = s_{ki} \hat{y}_{it} - (\hat{n}_i + \delta_t) k_{it} \tag{3.13}$$

Where s_{ki} is the proportion of income invested in physical capital and δ is the physical capital depreciation rate. $\hat{n}_i = n_i + g + \theta 1 q_i + \theta 2 h_i$

Following MRW (1992), we can show that (3.13) gives (3.14) and (3.15) at steady state:

$$k_i^* = \left[s_{ki} / (\hat{n}_i + \delta) \right]^{1/(1-\alpha)} \tag{3.14}$$

$$\hat{y}_i^* = \left[s_{ki} / (\hat{n}_i + \delta) \right]^{\alpha/(1-\alpha)} \tag{3.15}$$

Where the asterisk indicates the steady state value. The steady state values of output and capital per effective unit of labour are determined by the rate of investment in physical capital and the rate of growth of labour force, environment, education and technology.

Replacing (3.15) into (3.12), and using natural logarithms, we obtain (3.16).

$$\ln(\frac{Y_{it}^*}{L_{it}}) = \ln A_0 + gt - \frac{\alpha}{1-\alpha}\ln(\hat{n}_i + \delta) + \frac{\alpha}{1-\alpha}\ln(s_{ki}) + \theta 1 \ln(Q_{it}) + \theta 2 \ln(H_{it}) \quad (3.16)$$

The equation (3.16) shows that the investment in physical capital, human capital and natural environment improvement impacts positively the production per capita.

The variable Y^* cannot be observed since it supposes that we are at the steady state at the estimation period and this is a strong assumption. To solve for this problem, we use the linearization method of MRW (1992), Islam (1995), Bassanini and Scarpetta (2001 and 2007) and we have:

$$d \ln y_{it} / dt = -\lambda(\ln y_t - \ln y^*) \quad (3.17)$$

where $y = \frac{Y}{L}$ and $\lambda_t = (1-\alpha)(\hat{n}_i + \delta)$ is the speed of convergence. This speed of convergence changes with the addition of environmental variables through $\theta 1 q_i$, since $\hat{n}_i = n_i + g + \theta 1 q_i + \theta 2 h_i$. An improvement in environment quality increases the speed of convergence.

The transition dynamics through the steady state can be written as (3.18).

$$\ln y_t - \ln y_{t-s} = \psi(\ln y^* - \ln y_{t-s}) \quad (3.18)$$

Where (*t*-*s*) is a period arbitrary chosen.

Replacing steady state *y* value by it value in current period, (3.16) gives (3.19).

$$\begin{aligned} \ln(y_{it}) - \ln(y_{it-s}) = {} & \psi \ln A_0 + \psi g t - \frac{\alpha}{1-\alpha}\psi \ln(\hat{n}_i + \delta) + \frac{\alpha}{1-\alpha}\psi \ln(s_{ki}) \\ & + \psi\theta 1 \ln(Q_{it}) + \psi\theta 2 \ln(H_{it}) - \psi \ln(y_{it-s}) \end{aligned} \quad (3.19)$$

Where $\psi = (1 - \exp(-\lambda_i t))$

Equation (3.19) can be simplified by adding both $\ln(y_{t-s})$ to the left and right hand sides in order to have only $\ln(y_t)$ as left hand side member and we obtain (3.20).

$$\ln(y_{it}) = \psi \ln A_0 + \psi gt - \frac{\alpha}{1-\alpha}\psi \ln(\hat{n}_i + \delta) + \frac{\alpha}{1-\alpha}\psi \ln(s_{ki}) + \psi\theta 1 \ln(Q_{it}) + \psi\theta 2 \ln(H_{it}) + \exp(-\lambda t) \ln(y_{it-s}) \quad (3.20)$$

This equation (3.20) shows that environment quality is an important determinant of economic development.

5.4. Empirical analysis

5.4.1 Methodology

The analysis is subdivided into four main steps. First, the effect of environment quality on economic outcomes is assessed through the introduction of pollution indicators in an augmented neoclassical growth model. Then, we evaluate how these variables affect the ability of poor countries to catch up the rich ones by adding to the previous model the interaction term between initial gross domestic product (GDP) per capita and environmental variable. The third model investigates the role played by health in the impact of environmental variables on economic outcomes. Finally, we develop an explanation to this effect of pollution on convergence by estimating simultaneously a growth equation, a health equation, and an environmental equation to highlight the interrelationships between these three variables.

Economic growth and environment

Based on the neoclassical augmented growth model, the effect of environment on economic growth could be specified as follows:

$$\mathrm{gdpc}_{it} = \alpha_1 \, \mathrm{g}\, dpc_{it-1} + \alpha_2 envir_{it} + \alpha_k X'_{kit} + \upsilon_{it} \quad (4.1)$$

Where $gdpc_{it}$ and $envir_{it}$ represent respectively the logarithmic form of GDP per capita and the environment quality of country i in period t. X is the matrix of the control variables

including health introduced in the model, and which have been used frequently in the empirical literature.[23] ν_{it} is the error term. The coefficient of the economic catch up variable α_1 is expected to be superior to 0 and inferior to 1 ($0<\alpha_1<1$) according to the economic convergence hypothesis. We expect α_2 to be inferior to 0 ($\alpha_2<0$).

Using panel data, the econometric model takes into account countries specific effects and time-invariant heterogeneity. As we saw above, there is a reverse causality in the relationship between environment and economic outcomes. According to the Environmental Kuznets Curve hypothesis, the development level of a country has a significant effect on its level of pollution (Grossman & Krueger, 1995). Moreover, environmental indicator could also be a proxy of some variables that have a significant effect on economic growth, such as the technology use. The instrumental variable methods with the Two Steps Least Squares (2SLS) estimator might be appropriated in this case. But, this estimator applied to our model raises a problem because of its dynamic characteristics. Indeed it leads to a biased estimation of α_1 since $gdpcap_{it-1}$ and ν_{it} are correlated. The Generalized Method of Moments (GMM) applied for dynamic panel data is suitable to consistently estimate α_1 and also the coefficients of predetermined and endogenous variables. We use the System-GMM estimator which combines equation in levels and differences, and then exploits additional moment conditions (Blundell and Bond, 1998). Predetermined and endogenous variables are instrumented by their lagged values in levels and differences.[24] Two specification tests check the validity of the instruments. The first is the standard Sargan/Hansen test of over-identifying restrictions. The second test examines the hypothesis that there is no second-order serial correlation in the first-difference residuals.

[23] These variables are listed in the next subsection.

[24] The paper uses the two-step System-GMM estimator with the Windmeijer (2005) correction for finite sample bias.

The following model is then estimated:

$$\mathrm{gdpc}_{it} = \alpha_1 \, \mathrm{g}\, dpc_{it-1} + \alpha_2 envir_{it} + \alpha_k X'_{kit} + \mu_i + \kappa_t + \upsilon_{it} \qquad (4.2)$$

Where The country and time fixed effects are represented respectively by μ_i and κ_t.

Economic convergence and environment

To assess the impact of environmental quality on economic convergence, we introduce the interaction term between lag GDP per capita and environment as additional variable into the previous model.

$$\mathrm{gdpc}_{it} = \alpha'_1 \, \mathrm{g}\, dpc_{it-1} + \alpha_2 envir_{it} + \alpha_3 (\mathrm{g}\, dpc_{it-1}) * (envir_{it}) + \alpha_k X'_{kit} + \mu_i + \kappa_t + \upsilon_{it} \qquad (4.3)$$

In this model the catch up coefficient is $\frac{\partial(gdpc_t)}{\partial(gdpc_{t-1})} = \alpha'_1 + \alpha_3 * envir$ and this is a function of environmental quality. α'_1 is expected to be $0 < \alpha'_1 < 1$, $\alpha_2 < 0$ and $\alpha_3 > 0$.

Environment, health, and growth

The previous models allowed to assess the impact of environment degradation on economic growth and economic convergence when health status is among control variables. However, this remains insufficient because it does not take into account the interrelation between health, environment and economic growth. Moreover, it does not allow assessing the impact of environment degradation which affects growth through health. To assess this, we add to previous equation two other equations: an equation of health, and an equation of environment. Through these additional equations, we assess the impact of income and environmental degradation on health. Generally it is assumed that health outcomes of a population improve when the economy grows and this improvement are made easy by the rise in general standard of living (access to educational opportunities and health services). Health depends also on the

quality of physical environment such as the amount of air pollution and the quality of drinking water. At the same time, the quality of a country's physical environment is a result of certain growth factors in the economy (intensive use of land, forest, air and water pollution). The existing health theoretical models, including that of Grossman (1972) are not adapted to our econometric health equation. We follow Gangadharan and Valenzuela (2001) by expressing health as a function of income, physical environment quality and other control variables.

$$h_{it} = f(gdpc_{it}, envir_{it}(gdpc_{it}, z_{it}), w_{it}) \quad (4.4)$$

Where h is health indicator, z the non economic variables that determine environment quality and w the non economic variables that determine health status (provision and access to health services, physicians number, immunization rate, education). When we ignore the determinants of environment quality, the health equation can be written as (see Chapter 4):

$$h_{it} = \beta_0 + \beta_1 gdpc + \beta_2 envir_{it} + \beta_3 w_{it} + \rho_{it} \quad (4.5)$$

Here our purpose is to highlight the relation between economic development and environmental quality. Economic growth is generally made at the cost of a deterioration of the quality of the natural environment. But through which analytical relation development level affects environment? It is generally found that income is linked to environment quality through an inverted U relationship (EKC hypothesis). In our model environment quality is explained by income and some social variables.

$$envir_{it} = c + \gamma_1 gdpc_{it} + \gamma_2 gdpc_{it}^2 + \gamma_3 z_{it} + \eta_{it} \quad (4.6)$$

Where z is a vector of variables that could affect environment quality such as population density.

We estimate equations (4.2), (4.5) and (4.6) by the Three Steps Least Square method (3SLS). In addition to the explanation it brings to our results, the argument that guides this choice is the ability of this method to take into account the fact that the dependent variable of some equations can be used as explanatory variables in others. In fact in our system, the variable of

economic activity is both used as dependent variable and explanatory variable, it is the same for health and environment quality. This simultaneity bias can be corrected for each equation by the 2SLS method and for the system by the 3SLS.

5.4.2 Variables and data

This study is based on a panel data of 117 developed and developing countries for which data are available from 1971 to 2000 subdivided into five year periods.[25] The economic outcome is measured by GDP per capita based on purchasing power parity (PPP) in constant 2005 international dollars. This indicator is taken from the World Development Indicators (WDI 2008) of the World Bank. Environment quality is represented by three pollution indicators, carbon dioxide emission in metric tons per capita (CO2) and sulphur dioxide emission milligrams per GDP (SO2) for air pollution and Biological Oxygen Demand in milligrams per worker (BOD) for water pollution. The CO2 is a global measure of air pollution and affects health mainly through climate change induced diseases (Diarrhoeal diseases, malaria, selected unintentional injuries, protein-energy malnutrition). The SO2 is a local air pollutant and affects population health through Respiratory infections, selected cardiopulmonary diseases, lung cancer. BOD is a measure of the oxygen used by micro-organisms to decompose waste. Micro-organisms such as bacteria are responsible for decomposing organic waste. When organic matter such as dead plants, leaves, grass clippings, manure, sewage, or even food waste is present in a water supply, the bacteria will begin the process of breaking down this waste. If there is a large quantity of organic waste in the water supply, there will also be a lot of bacteria present working to decompose this waste. In this case, the demand for oxygen will

25 The time periods are1971-1975 ; 1976-1980 ; 1981-1985 ; 1986-1990 ; 1991-1995 ; 1996-2000.

be high (due to all the bacteria) so the BOD level will be high (CIESE[26]). The BOD degrades health outcomes through Diarrhoeal diseases, trachoma, schistosomiasis, ascariasis, trichuriasis, hookworm disease. The BOD and CO2 are also taken from WDI 2008 while Sulfur dioxide emission (SO2) is from the dataset compiled by David Stern[27] in 2004. The health indicator used is the infant mortality rate (IMR). Infant mortality is preferred in this Chapter because of data availability, since the DALYs are not available at country level for a time longue period. As this indicatorr is limited asymptotically, and an increase in this indicator does not represent the same performance when its initial level is weak or high, the best functional form to examine is that where the variable is expressed as a logit, following Grigoriou (2005), we use the logistic form of this variable.

$$\log it(IMR) = \log(\frac{IMR}{1-IMR}).$$

We use as control variables in the growth equation (Equation 4.2), the investment ratio measured by the Gross Fixed Capital Formation as percentage of GDP, human capital accumulation (education), and annual population growth rate. These variables are traditionally used in the empirical assessment of growth determinants (see Mankiw et al, 1992) and largely discussed in the theoretical model. We also control for trade openness (ratio of the sum of import and export to GDP), household final consumption per capita, financial development (money and quasi money as a ratio of GDP), institutions quality, and inflation rate to capture macroeconomic stability, and policy information.

The variables that explain health (Equation 4.5) are the economic development level, the supply of health services and infrastructures (immunization rate against DPT, the number of

[26] According to the Center for Improved Engineering and Science Education (CIESE): http://www.k12science.org/curriculum/waterproj/bod.shtml

27 We thank David Stern for the provision of data

physicians per 1000 inhabitants), and demographic variables (women fertility rate, and the percentage of urban population).

Finally, with regard to the environmental equation, we use as determinant of environmental pollution, the GDP per capita and GDP per capita squared to verify the EKC hypothesis, Income inequality (measured by the Gini coefficient), and demographic variables (women fertility rate, and the percentage of urban population).

All these indicators are taken from World Bank World Development Indicators (WDI 2008) apart from our institutions quality indicator taken from polity IV, and the variable used is polity2; the variable of education quality from Barro and Lee 2000; and the income inequality indicator taken from the database created by Galbraith and associates and known as the University of Texas Inequality Project (UTIP) database. The descriptive statistics, the definitions and the sources of these variables as well as the list of countries are respectively presented in the appendix 5.2, appendix 5.3 and appendix 5.4.

5.5. Econometric results

We begin by discussing the results from the estimation of the growth model, then, we present the results obtained with the simultaneous estimation of the three equations.

5.5.1 Economic growth and the environment

The results obtained from the estimation of equation 4.2 are presented in the first three columns of Table 5.1. The dependent variable is GDP per capita and our variable of interest is environment quality, measured by three different indicators (SO2 per GDP, CO2 per capita and BOD per worker). This equation is estimated with the two-steps System-GMM estimator and environmental variables are taken as endogenous and then instrumented by at least their

second order lags.[28] Results suggest that environmental degradations have a negative and statistically significant effect on economic growth whatever the environmental indicator considered, infant mortality has a negative and significant effect on economic growth. A 1% increase in SO2, CO2, and BOD emissions provokes a reduction of economic growth rate of 0.622, 0.007, and 0.666 points of percentage respectively.

Another interesting result is the coefficient of the catch up variable. Indeed, the coefficient of lagged GDP per capita is around 0.91, this corresponds to a rate of convergence of about 2% per year. That means that, each year poor countries reduce their gap to their steady state to 2 percent. This convergence rate is closed to that found in the literature. Health incator appears with a negative and significant coefficient, showing the negative effect of infant mortality on economic growth as in Chapter 4.

Regarding the control variables, only investment, institutions quality and inflation rate appear statistically significant. In fact, investment, trade openness and institution quality increase economic growth while inflation rate reduces it.

[28] To prevent the problem of the proliferation of instruments commonly faced in this methodology, we restrict the maximum number of lags at 5, what leads us to a maximum number of instruments equal to 26.

Table 5. 1: Two-step System-GMM results of the effect of environmental variables on Economic growth and convergence

	Dependent variables: GDP per capita PPP in constant value 2005					
VARIABLES	SO2 per GDP (1)	CO2 per capita (2)	BOD per worker (3)	SO2 per GDP (4)	CO2 per capita (5)	BOD per worker (6)
Log Initial GDP per capita	0.913***	0.917***	0.907***	0.903***	0.936***	0.675***
	(14.73)	(8.73)	(42.12)	(13.40)	(5.19)	(6.74)
(Environment)x(Initial GDP)				2.313**	0.013***	0.910**
				(2.36)	(2.98)	(2.40)
Environmental variables	-0.622**	-0.007*	-0.666*	-16.547**	-0.128***	-7.692**
	(2.00)	(1.93)	(1.66)	(2.36)	(2.94)	(2.42)
Population growth	-0.000	0.003	-0.008	0.001	-0.002	0.006
	(0.06)	(0.53)	(0.99)	(0.33)	(0.26)	(0.53)
Log Schooling	0.013*	0.005	0.011	0.005	0.002	0.014
	(1.94)	(0.45)	(1.16)	(0.75)	(0.19)	(1.07)
Log Investment rate	-0.015	0.091***	0.051	0.090***	0.134***	0.064*
	(0.44)	(3.68)	(1.64)	(3.26)	(3.36)	(1.85)
Health	-0.048***	-0.044***	-0.028*	-0.040***	-0.035***	-0.080***
	(4.03)	(4.15)	(1.77)	(3.26)	(2.66)	(2.63)
Openness	0.056**	0.018	0.037	0.023	0.018	-0.036
	(2.32)	(0.75)	(1.53)	(1.46)	(0.72)	(0.95)
Log Consumption	0.049	0.050	0.043**	0.041	0.018	0.078
	(0.88)	(0.59)	(2.36)	(0.76)	(0.13)	(1.15)
Financial development	-94.851	-66.054	-132.090***	-83.703	-102.375	151.914
	(1.25)	(1.41)	(2.95)	(1.19)	(1.60)	(1.37)
polity2	0.001	0.002**	0.002**	0.003***	0.002**	0.002*
	(1.31)	(2.21)	(1.98)	(2.76)	(2.17)	(1.72)
inflation	0.005*	-0.003***	-0.003***	-0.002***	-0.003***	-0.002***
	(1.72)	(5.44)	(5.91)	(5.18)	(3.70)	(2.60)
Constant	0.228	-0.066	0.357*	0.106	-0.067	1.732***
	(1.31)	(0.30)	(1.93)	(0.69)	(0.17)	(2.85)
Observations	235	239	203	235	239	203
Countries	68	69	63	68	69	63
AR1	0.019	0.009	0.014	0.004	0.010	0.010
AR2	0.127	0.094	0.117	0.128	0.115	0.151
Hansen p-value	0.388	0.156	0.259	0.389	0.285	0.139
Number of instruments	26	17	15	17	17	19

Note: Robust t-statistics in parentheses. Standard errors are corrected by the Windmeijer (2005) method designed for finite sample bias in a two-step System-GMM estimator. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

5.5.2 Economic convergence and environment quality

To assess empirically whether pollution affects the speed of convergence, equation 4.3 is estimated under the hypothesis that environmental variables and the interaction term are endogenous. As previously argued, environmental degradation may reduce the ability of poorer countries to catch up richer ones. The results obtained are summarized in the last three columns (4, 5 and 6) of Table 5.1. The coefficients of our variables of interest have the correct signs and are statistically significant. Indeed, the lag of GDP per capita and its interaction term with environmental indicators have positive coefficients, while pollution variables have negative coefficients. This means that the speed of convergence of an economy depends on its pollution level. More precisely, a high level of environmental degradation increases the marginal effect of lag GDP per capita on its current level and therefore reduces the speed of convergence. Environment quality can be viewed as an obstacle for developing countries by reducing their ability to get closer to developed countries economically, given the Environmental Kuznets Curve hypothesis.

The scarcity of education data reduces the number of countries in our sample, since it is not available for many countries. In order to control for the robustness of our results, we estimated our models without the education variable. The results are presented in Table 5.2.

The sample size increases from 68 countries to 86 and the results remain unchanged, namely, environmental degradation affects the ability of poor economies to cach up developed ones.

Table 5. 2: Two-step System-GMM results of the environmental variables effect on Economic convergence without education

	Dependent variables: GDP per capita PPP in constant value 2005		
	SO2 per GDP	CO2 per capita	BOD per worker
VARIABLES	(1)	(2)	(3)
Log Initial GDP per capita	0.891***	0.870***	0.797***
	(10.59)	(5.83)	(12.29)
(Environment)x(Initial GDP)	1.520*	0.010*	0.690*
	(1.66)	(1.94)	(1.94)
Environmental variables	-11.060*	-0.105*	-5.832*
	(1.69)	(1.94)	(1.96)
Population growth	-0.000	-0.003	-0.001
	(0.07)	(0.38)	(0.11)
Log Investment	0.068**	0.124***	0.056*
	(2.28)	(2.81)	(1.92)
Health	-0.031***	-0.014	-0.050**
	(2.71)	(0.84)	(2.47)
Openness	0.031	0.067*	-0.013
	(1.27)	(1.79)	(0.40)
Log Consumption	0.055	0.078	0.015
	(0.78)	(0.67)	(0.54)
Financial development	-45.268	-131.795*	103.831
	(0.76)	(1.72)	(1.10)
polity2	0.002**	0.002	0.002*
	(1.99)	(1.63)	(1.74)
inflation	-0.003***	-0.002***	-0.003***
	(5.88)	(3.73)	(7.03)
Constant	0.214	0.131	1.315**
	(1.19)	(0.35)	(2.18)
Observations	287	292	233
Countries	84	86	73
AR1	0.006	0.017	0.003
AR2	0.129	0.150	0.106
Hansen p-value	0.191	0.210	0.545
Number of instruments	13	18	14

Note: Robust t-statistics in parentheses. Standard errors are corrected by the Windmeijer (2005) method designed for finite sample bias in a two-step System-GMM estimator. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

5.5.3 Interrelationships between income, health and environment

To take into account the interrelationships between health, environment and economic growth, and to assess the impact of environmental degradation which affects growth through health, we estimate simultaneously equations (4.2), (4.5) and (4.6) with the Three Steps Least Squares (3SLS) estimator. The results obtained are presented in table 5.3.

The first three columns of this table (columns 1, 2 and 3) present the results when sulphur dioxide per GDP (SO2) is used as environmental indicator. These results show that environmental degradation and mortality rate (as in Chapter 4) reduce economic growth (Column 1). GDP per capita, immunisation rate, and physicians number are factors that contribute to improve health status, while environment degradation and fertility rate worsen it (Column 2). The positive coefficient of environment variable combined with the negative effect of health on growth does not rejet our theoretical argument, namely health is an important channel through which pollution affects economic growth. The results of the environmental quality equation in column 3 indicate that the coefficient of income per capita is positive and significant at 1%, showing that economic activity deteriorates environment quality. But the negative and significant coefficient of income square indicates that the negative effect of GDP on environment quality is conditioned to an income threshold above which the effect becomes positive and income improves environment quality confirming the Environmental Kuznets Curve hypothesis (EKC). The six last columns of this table present the results when carbon dioxide per GDP (columns 4, 5 and 6) and the biological oxygen demand (columns 7, 8 and 9) are used as environmental variables. The environmental variables have the expected sign and the EKC hypothesis is verified in each case.

Table 5. 3: 3SLS estimation of the interrelationships between health, environment and economic

	3SLS estimation of the relationships between health, environment and economic activity								
VARIABLES	GDP per capita (1)	Inf. Mort. (2)	SO2 per GDP (3)	GDP per capita (4)	Inf. Mort. (5)	CO2 per capita (6)	GDP per capita (7)	Inf. Mort. (8)	BOD per worker (9)
Pop. growth	-0.00696			0.00187			-0.00570		
	(-1.197)			(0.408)			(-1.008)		
Initial GDP	0.920***			0.979***			0.905***		
	(54.32)			(29.17)			(67.29)		
Schooling	0.0245***			0.0244***			0.0244**		
	(2.900)			(3.214)			(2.545)		
Investment	0.0884***			0.113***			0.0454**		
	(5.016)			(4.898)			(2.007)		
Health	-0.091***			-0.154***			-0.0897***		
	(-4.594)			(-7.337)			(-3.033)		
Log Cons.	-0.00927			-0.00996			0.0269*		
	(-0.393)			(-0.593)			(1.869)		
Financial dev.	-129.5			-17.75			-28.18		
	(-1.473)			(-0.473)			(-0.584)		
polity2	0.00119			0.000830			0.00203***		
	(1.322)			(1.143)			(2.826)		
inflation	-0.000972			-0.00229			-0.00214		
	(-0.630)			(-1.418)			(-1.383)		
Immunization		-0.85***			-0.33***			-0.49***	
		(-5.272)			(-2.729)			(-3.417)	
Physician		-0.0789*			-0.0596			-0.13***	
		(-1.951)			(-1.577)			(-3.014)	
Fertility rate		0.645***			0.925***			0.602***	
		(6.697)			(8.245)			(5.263)	
Environment	-0.069***	0.465***		-0.0550**	0.458***		-0.0992	0.845***	
	(-3.180)	(6.216)		(-2.568)	(5.114)		(-1.281)	(3.298)	
GDP per		-0.19***	4.045***		-0.94***	4.455***		-0.35***	0.308
		(-3.520)	(5.464)		(-9.285)	(7.825)		(-7.731)	(1.615)
(GDP per capita) ^{2}			-0.26***			-0.18***			-0.0213*
			(-6.089)			(-5.588)			(-1.875)
inequality			-0.00165			-0.005			0.0125***
			(-0.169)			(0.60)			(4.511)
Constant	-0.252**	0.887*	-20.3***	-0.615**	3.655***	-23.3***	-0.0273	0.832	-3.202***
	(-2.518)	(1.755)	(-6.573)	(-2.291)	(4.684)	(-9.858)	(-0.353)	(1.371)	(-4.087)
Observations	179	179	179	216	216	216	180	180	180
R-squared	0.993	0.724	0.197	0.994	0.798	0.817	0.997	0.840	0.262

Note : Robust t-statistics in parentheses. $^{*}\ p < 0.10$, $^{**}\ p < 0.05$, $^{***}\ p < 0.01$. All the independent variables are in natural logarithmic form, except health variable, population growth, polity2 and inflation rate.

The 3SLS estimations of these three equations allow us to draw some conclusions: there is an inverse causality between economic activity and environmental degradation and health status is an important channel through which environment degradation affects economic growth even if it is not alone. The effect of economic activity on environment quality being dependent on income level, countries whose income is below the EKC income threshold will slow down in a poverty trap due to environment degradation. However, those whose income is above this threshold will be in a virtuous circle due to the improvement of environment quality. This could reduce the ability of poor countries to catch up the rich ones. Any ambitious economic policy must take into account environmental concerns to avoid it perverse effects.

5.6. Concluding remarks

The main goal of this chapter is the analysis of the interrelationships between health, income and environment degradation, and its consequences on economic convergence process. We introduce environment variable in a growth model and we measure econometrically its effect on economic growth. Our results show that environmental degradation negatively affects economic activity and reduces the ability of poor countries to reach their steady state. This reinforces our theoretical argument according to which environment quality improvement plays a considerable role in economic convergence process. Two-steps GMM and Least square estimations of growth, environment, and health equations allow us to verify the inverse causality between environment quality and economic growth and between economic growth and health. Health status remains an important channel through which environment degradation affects economic growth even if it is not alone. Poor countries which have chosen rapid economic growth at the price of environment quality will penalise themselves and have

little chance to reach their goal. Such policy can reduce growth through health and other channels.

Poor countries cannot postpone attending environmental concerns in the hope that the environment will improve with increased incomes and avoid poverty trap due to environment degradation. Policy makers in these countries should in contrary take into account environmental concerns as promoted by international community through the MDGs.

This chapter can also be placed into the debate about development aid effectiveness. In fact, a development assistance based on less polluting production technology will help poor countries to avoid the vicious circles shown in this chapter.

One way this research can be extended is to use other health and environment indicators and compare the results for each indicator. Another way to extend this chapter is the use of other technical approach in order to confirm our idea.

APPENDICES 5

Appendix 5.1: Classification of macroeconomic models of environmental degradation and growth

groups	characteristics	models	remarks
Optimal growth models build on a Ramsey (1928), Koopmans (1960) and Cass (1965).	These are dynamic optimization models, in which the utility-maximization problem of the infinitely lived consumer is solved using the techniques of optimal control theory. Either the stock or the flow of pollution is an argument of both the production function and the utility function of the representative consumer. These models extend the basic dynamic optimization of Ramsey, Cass and Koopmans to include the disutility of pollution that arises as a result of economic activity.	Brock and Taylor (2004), Keeler et al. (1971), D'Arge and Kogiku (1973), Mäler (1974), Forster (1973), Gruver (1976), Brock (1977), Becker (1982), Tahvonen and Kuuluvainen (1994), Selden and Song (1995), Stokey (1998)	Provide in general of theoretical foundation for the empirically observed Environmental Kuznets Curve, and the Steady state level of consumption and capital accumulation is lower than the steady state consumption and capital accumulation of a model without pollution
Models of the Environment as a Factor of Production	In addition to the use of pollution as an argument of the production and utility function these models include the environment itself. Environment is interpreted as a stock natural capital that the economy is endowed with or the aggregate level of environmental	Lopez (1994), and Chichilinsky (1994).	Property rights are decisive in determining whether environmental degradation eventually declines with growth.

	quality.		
Endogenous Growth Models of Environmental Degradation and Growth.	Inclusion of the environment or pollution as a factor of production and environmental quality as an argument of the utility function Endogenous Growth Models of Romer (1986, 1990), Barro (1990), Robelo (1991) and Lucas (1988).	Bovenberg and Smulders (1995, 1996), Ligthhard and van der Ploeg (1994), Gradus and Smulders (1993), Stokey (1998), Hung, Chang, and Blackburn (1994)	The conclusions are similar to the ambiguous predictions of the neo-classical growth models because the results again depend on the form of the utility function.
Other Macroeconomic Models of Environmental Degradation and Growth	Several other macroeconomic models of environmental degradation and growth different from those already discussed could be found.	The many-goods general equilibrium model with two regions (North and South) of Copeland and Taylor (1994) and the overlapping generation's model of John and Pecchenino (1994, 1995) based on Diamond (1965).	

Source : Author based on Panayotou (2000).

Appendix 5.2 : Descriptive Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
GDP per capita	259	11212.43	10918.89	355.8692	55491.52
Inf. Mort. rate	259	36.90442	33.55625	3.48	138.656
SO2 per GDP	253	0.0069203	0.017175	0.0000922	0.1760821
CO2 per capita	259	5.060414	5.543132	0.0319344	35.87007
BOD per worker	256	0.1950967	0.0519381	0.0694487	0.4478187
Pop. growth	259	1.337404	3.075527	-44.40836	5.603235
school	211	23.11564	22.01362	0	84.1
investment	258	20.90701	5.34708	9.488747	40.29905
openness	256	68.85741	39.29941	2.003065	238.6728
consumption	219	4469.355	5270.451	87.23995	22281.84
Financial Dev.	221	44.7538	32.07666	9.198633	227.4642
polity2	226	3.879646	6.691901	-10	10
Inflation rate	254	38.59134	190.1751	-1.659683	2342.221
Immunization	259	81.51004	16.49692	24	99
Physician	259	1.445306	1.155825	.0198895	4.173381
Fertility rate	259	3.132003	1.578447	1.152	7.845
inequality	259	42.36337	6.444149	26.135	64.2473

Appendix 5.3 : Variables definitions and sources

Variables	characteridtics	sources
GDP per capita	gross domestic product per capita	WDI 2008
Inf. Mort. rate	infant Mortality rate	UNICEF
SO2 per GDP	sulphur dioxide emission per GDP	David Stern
CO2 per capita	Carbon dioxide emission per capita	WDI 2008
BOD per worker	Biological Oxygen Demande per worker	WDI 2008
Pop. growth	population growth rate	WDI 2008
school	Percentage of "no schooling" in the total population	Barro and Lee 2000
investment	gross fixed capital formation	WDI 2008
openness	Ratio of the sum of export and import to GDP	WDI 2008
consumption	Household final consumption rate per capita	WDI 2008
Financial Dev.	Money and quasi money as a ratio of GDP	WDI 2008
polity2	institution quality	polity IV
Inflation rate	consumption index price	WDI 2008
Immunization	immunization rate against DPT	WDI 2008
Physician	number of physicians per 1000 inhabitants	WDI 2008
Fertility rate	women fertility rate	WDI 2008
inequality	gini coefficient of income	university of Texas income inequality

Appendix 5.4 : list of countries in the sample

Country name	Country name	Country name
Albania	Greece	Norway
Argentina	Guatemala	Nepal
Armenia	Honduras	New Zealand
Australia	Croatia	Oman
Austria	Haiti	Pakistan
Azerbaijan	Hungary	Panama
Belgium	Indonesia	Peru
Bangladesh	India	Philippines
Bulgaria	Ireland	Papua New Guinea
Bahrain	Iran, Islamic Rep.	Poland
Belize	Iceland	Portugal
Bolivia	Israel	Paraguay
Brazil	Italy	Romania
Bhutan	Jamaica	Russian Federation
Botswana	Jordan	Rwanda
Central African Republic	Japan	Saudi Arabia
Canada	Kenya	Senegal
Chile	Kyrgyz Republic	Singapore
China	Korea, Rep.	El Salvador
Cote d'Ivoire	Kuwait	Suriname
Cameroon	Sri Lanka	Slovak Republic
Congo, Rep.	Lithuania	Slovenia
Colombia	Luxembourg	Sweden
Cape Verde	Latvia	Swaziland
Costa Rica	Morocco	Syrian Arab Republic
Cyprus	Moldova	Thailand
Germany	Madagascar	Tonga
Denmark	Mexico	Trinidad and Tobago
Algeria	Macedonia, FYR	Tunisia
Ecuador	Malta	Turkey
Egypt, Arab Rep.	Myanmar	Uganda
Spain	Mongolia	Ukraine
Ethiopia	Mozambique	Uruguay
Finland	Mauritius	United States
Fiji	Malawi	St. Vincent and the Grenadines
France	Malaysia	Venezuela, RB
Gabon	Namibia	South Africa

United Kingdom Ghana	Nigeria Netherlands	Zambia

General Conclusion

The achievement of the millennium development goals adopted by the United Nations in September 2000 by the target date 2015 remains an important concern. Therefore, all the studies and efforts aiming to reduce poverty rate, to improve population's health and physical environmental quality are welcome. It is commonly recognized by scholars, policy makers as well as the international community that better health outcome is an important predictor of economic development and poverty reduction, since it increases the productivity and the availability of the labour force, and the accumulation of physical capital. It is also largely accepted and documented that sustainable economic development requires clean physical environment, and economic growth is an important determinant of environmental quality. But, less attention has been given to the simultaneous association among human capital, natural capital, and economic development.

This dissertation bridged this gap by analyzing theoretically as well as empirically the interrelationships among population's health, environmental degradation and economic development, its consequences for developing countries, and some effective policy responses. Four important issues are theoretically and empirically addressed in this dissertation: (i) What role does environmental degradation play in the association between income distribution and health? (ii) Is environmental degradation a determinant of the large health inequality observed between and within developing countries? (iii) Does the global burden of disease matter for economic development? (iv) How do the interrelationships among health, environmental quality and economic development affect the ability of poor countries to converge towards developed ones economically?

The first part answered to the first two questions through chapter 2 and chapter 3. The second chapter analyzed whether environmental degradation could be considered as an additional channel through which income inequality affects infant and child mortality. The third chapter

analyzed the role played by the degradation of air quality in health inequalities between and within developing countries, and the role of political institutions in this relationship.

Indeed, the theoretical and empirical results show that income inequality affects negatively environmental quality and environment degradation worsens population's health. Another interesting result is that air and water pollutions are important channel through which income inequality affects population health.

The chapter 3 showed that the large inequalities in infant and child mortality rates between and within developing countries are partly attributable to air pollution. More precisely, population belonging to poorest income quintiles are those likely to suffer more from environmental degradation, because they receive the highest exposure, and this exposure then exercises larger effects on their health than it does on the average population. Furthermore, richest communities have more prevention than the poorest and have more access to medical care when they are sick from pollution. Another interesting finding of this chapter is the role of political institutions in this relationship. In countries with good political institutions, this heterogeneity in the health effect of pollution is reduced since these institutions favour universal health policy issues, information and advices about hygiene, and health infrastructures building.

The second part of the dissertation responded to the two last questions (chapter 4 and chapter 5). The chapter 4 assessed empirically the effect of health (global burden of disease, communicable disease, and malaria) on economic growth. The interrelationships among health, environment, and economic development as well as the consequences of these interrelationships on the convergence of poor countries towards developed countries are investigated theoretically and empirically in the last Chapter.

The investigation of the role of health outcomes in economic growth (chapter 4) showed that health indicators, when correctly measured by the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability, and when accurately instrumented, have significant impact on economic performance unlike the recent works of Acemoglu and Robinson (2009). This important effect of health status in the economic development process holds for more aggregate health outcomes as well as less aggregated health measurement (communicable diseases and malaria), and for the overall sample of countries as well as the sample of developing countries. This is particularly more important for developing world where health situation is catastrophic. Indeed, improving health situation could be considered as an opportunity since it will give them enough rooms to foster economic performance and reduce poverty level.

The examination of the interrelationships among population's health, environmental degradation and economic development (chapter 5) pointed out important results. Theoretically as well as empirically, we found that water pollution and the degradation of air quality negatively affect economic performance. Moreover, the Environmental Kuznets Curve (EKC) hypothesis is verified, namely, environmental degradation rises faster in the early stages of economic development, then slows down, reaches a turning point and declines with further income growth. Health status remains an important channel through which environment degradation affects economic growth even if it is not alone. Another important finding is that, environment degradation reduces the ability of poor countries to reach developed one economically. This reinforces our argument according to which environment quality improvement plays a considerable role in economic convergence process.

These results call for important policy implications. First, there are important interrelationships among the MDGs adopted by the United Nations in September 2000, therefore efforts might be focused simultaneously on all of them instead of making a

hierarchy among them. Making choices among them and postponing some to the future will create a kind of "weak link" and prevent the achievement of all the targets. Health preoccupations (Goals 4, 5 and 6) are important determinants of poverty reduction (Goal 1) through economic growth and it is difficult to improve health status without improving the income level.

Moreover developing countries cannot postpone attending environmental concerns (Goal 7) in the hope that the environment will improve with increased incomes and avoid poverty trap due to environment degradation. This behaviour could seriously constrain their performance. Policy makers in these countries should in contrary take into account environmental concerns as promoted by the international community through the MDGs.

Environmental quality could be improved using the existing environmental friendly technologies, and research and development (R&D) programmes. This will help developing countries to innovate and encourage learning by doing, which is important to reduce abatement cost. Moreover the effectiveness of development aid should take into account environmental concerns. In fact, a development assistance based on less polluting production technology will help poor countries to avoid the vicious circles shown in this dissertation.

Another important policy implication comes from chapter 2. Given that developing countries are characterized by high income inequality, their government may implement distributive policy in order to avoid its negative impact on environment and health. Moreover, income distribution is an important predictor in the achievement of the Millennium Development Goals. The international community as well as governments should pay more attention to the consequences of their policies on income inequality in order to improve health outcomes and physical environment quality.

Finally, to be effective, health policies should not be based only on average health of a given population, but also on its distribution. In addition, differential distribution of health effects of pollution should be considered alongside differential distribution of the benefits related to the emission sources. Indeed, those who pollute more in a population, such as car ownership may compensate those who bear the adverse effect by paying a tax. Moreover, improving political institutions is not only important for economic growth, but it is also essential for population wellbeing.

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Contents

List of Tables

List of Figures

Résumé de la thèse

Cette thèse étudie théoriquement et empiriquement les interrelations entre la santé de la population, la dégradation de l'environnement et le développement économique, ses conséquences pour les pays en développement, et fournit certaines réponses en termes de politique économique. Elle est subdivisée en deux parties. La première partie s'intéresse à la relation entre l'environnement, la santé, et les inégalités. Elle analyse dans un premier temps l'hypothèse selon laquelle la dégradation de l'environnement pourrait être considérée comme un canal supplémentaire par lequel les inégalités de revenu affectent les taux de mortalité infantile et juvénile (chapitre 2). Nos travaux théoriques et empiriques montrent que les inégalités de revenu affectent négativement la qualité de l'air et de l'eau, et cela à son tour dégrade la santé de la population. Par conséquent, la dégradation de l'environnement peut être considérée comme un canal non négligeable à travers lequel les inégalités de revenu influence l'état de santé. Il est ensuite démontré que les émissions de dioxyde de soufre (SO2) et celles des micro-particules (PM10) sont en partie responsables des grandes disparités dans la mortalité infantile et juvénile au sein des pays pauvres (chapitre 3). En outre, nos résultats soutiennent l'idée selon laquelle les institutions démocratiques jouent un rôle de protection sociale en atténuant cet effet pour les classes de revenu les plus pauvres et ainsi réduisent les inégalités de santé provoquées par la pollution. La deuxième partie évalue le lien entre la santé, l'environnement et la croissance économique. Le Chapitre 4 évalue l'effet de la santé (charge globale de la maladie, maladies transmissibles et paludisme) sur la croissance économique. Ce chapitre montre que les indicateurs de santé, lorsqu'ils sont correctement mesurés par l'écart entre l'état de santé actuel et une situation de santé idéal où toute la population vit à un âge avancé, indemne de maladie et d'invalidité, et lorsqu'ils sont convenablement instrumentés, ont un impact négatif significatif sur la performance économique. Les conséquences de ces interactions sur la convergence économique des pays pauvres vers leur état régulier, sont théoriquement et empiriquement analysées dans le dernier chapitre. Il en ressort que la dégradation de l'environnement réduit la capacité des pays pauvres d'atteindre leur état regulier, renforçant ainsi notre argument théorique selon lequel l'amélioration de la qualité de l'environnement joue un rôle considérable dans le processus de convergence économique. En outre, la dégradation de la qualité de l'air et de l'eau affecte négativement la performance économique, et l'état de santé demeure un canal important par lequel la dégradation de l'environnement agit sur la croissance économique même si elle n'est pas le seul. L'hypothèse de la courbe environnementale de Kuznets (EKC) est également vérifiée.

Summary of the thesis

This dissertation investigates theoretically and empirically the interrelationships among population's health, environmental degradation and economic development, its consequences for developing countries, and some effective policy responses. The first part explores the association between health, environment, and inequalities. It firstly analyzes whether environmental degradation could be considered as an additional channel through which income inequality affects infant and child mortality (chapter 2). Theoretical and empirical investigations show that income inequality affects negatively air and water quality, and this in turn worsens population's health. Therefore, environmental degradation is an important channel through which income inequality affects population health. Then, it is shown that sulphur dioxide emission (SO_2) and particulate matter (PM_{10}) are in part responsible for the large disparities in infant and child mortalities between and within developing countries (chapter 3). In addition, we found that democratic institutions play the role of social protection by mitigating this effect for the poorest income classes and reducing the health inequality it provokes. The second part is devoted to the link among health, environment, and economic growth. The effect of health (global burden of disease, communicable disease, and malaria) on economic growth is assessed in Chapter 4. This chapter shows that health indicators, when correctly measured by the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability, and when accurately instrumented have significant impact on economic performance. The consequences of these interrelationships on the convergence of poor countries towards their steady state are theoretically and empirically investigated in the last Chapter (chapter 5). It is found that environment degradation reduces the ability of poor countries to reach their own steady state, reinforcing our argument according to which environment quality improvement plays a considerable role in economic convergence process. Moreover, the degradation of air and water quality affects negatively economic performance, and health status remains an important channel through which environment degradation affects economic growth even if it is not alone. The Environmental Kuznets Curve (EKC) hypothesis is also verified.

Keywords: Disease Global Burden, DALYs, economic growth, Environmental quality, Health indicator, economic convergence, income inequality, instrumental variables method, health inequality, air pollution, political institutions, social protection